ENDANGERMENT ASSESSMENT FOR ENVIRONMENTAL CONSERVATION AND CHEMICAL CORPORATION SITE

ZIONSVILLE, INDIANA

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TABLE OF CONTENTS

TABLE OF CONTENTS
(continued)

TABLE OF CONTENTS
(continued)

LIST OF TABLES

LIST OF TABLES
(continued)

LIST OF FIGURES

EXECUTIVE SUMMARY

This Endangerment Assessment shows that risk from contaminants at the ECC site under a "no action" scenario are within the range normally found acceptable by USEPA at Superfund sites. The hazard indices for subchronic exposure and noncarcinogenic chronic exposure are shown to be less than 1 for on-site or off-site populations. Carcinogenic risk is shown to be less than $1x10^{-4}$.

This Endangerment Assessment (EA) of the ECC site has been prepared by the ECC Settlers as an alternate to the EA prepared by the USEPA, as part of the RI/FS. Data presented in the ECC RI/FS documents which have been reevaluated consistent with review comments prepared by the ECC Settlers are the basis for this assessment.

Two (2) significant assumptions were made which differ from those made in the original ECC-RI. First, because of low yield characteristics, the glacial till saturated zone was assumed not to be sufficient to support a water supply source for residential housing on the site. Secondly, decay of both the mass of source contaminants as well as those contaminants traveling from that source was assumed to occur in accordance with generally accepted physico-chemical behavior for constituent compounds.

The largest portion of calculated risk is due to presumed dermal absorption and ingestion of on-site soils. Little or no risk is associated with off-site migration and exposure. These conclusions suggest that simple access restriction to the site would substantially reduce risk exposure below those levels determined herein. In summary, if access restrictions are implemented, endangerment from the ECC site is effectively eliminated.

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1.0 INTRODUCTION

1.1 Purpose of CERCLA Endangerment Assessments

The need to include estimates of risk in the decision making process for contaminated sites has been recognized by the U.S. Environmental Protection Agency (USEPA), and is now a required part of the Comprehensive Environmental Response Compensation and Liability Act (CERCLA) RI/FS process. An endangerment assessment evaluates the demographic, geographic, physical, chemical, and biological factors at a site to determine whether there is a risk to public health or the environment.

The process can be used to evaluate the current risk as well as the risk that would be mitigated by implementation of alternative remedial actions. Thus, quantitatively derived estimates of risk may be used to determine if present conditions pose a health/environmental threat, and what effect on that risk various remedial actions might have.

1.2 Objectives

This Endangerment Assessment (EA) evaluates the level of risk posed to human populations and the environment as a result of the following modes of contaminant transport:

- volatilization from contaminated soil and ground water to the air,

- release from contaminated soils through percolation of precipitation and/or movement of ground water, and

- release from contaminated soil and ground water to surface water through direct ground water discharge.

In addition, the following exposure scenarios are considered:

- future use of the site as a residential area

- recreational use of the site

The assessment considers risks from potential and accepted carcinogens and non-carcinogens and compares concentrations of contaminants under current and predicted future conditions with potential applicable and relevant environmental standards. Risks evaluated in this assessment should be considered in light of remedial alternatives discussed in the ECC Feasibility Study and ECC/NSL Combined Alternatives Analysis.

2.0 METHODOLOGY

2.1 USEPA's Endangerment Assessment Process for CERCLA Sites

The purpose of this section is to provide an outline of the CERCLA Endangerment Assessment (EA) process. This outline forms the basis of this EA for ECC. The discussion is not intended to be a comprehensive guide to preparing risk assessments. Guidelines have been proposed for the preparation of EA's by the USEPA. These draft/proposed guidelines include the Draft Endangerment Assessment Handbook (USEPA, 1985a), Superfund Public

Health Manual (USEPA, October 1986), and Toxicology Handbook (USEPA, 1986b).

An EA is normally conducted after the completion of the Remedial Investigation (RI) field work as part of the RI/FS process. The RI field work determines the nature and extent of contamination at a site, and its results form the data base on which potential exposures can be determined and risks assessed. In addition, the RI defines the potential for contaminant movement from the site.

There are four evaluations which must be completed in a CERCLA EA (Figure 1):

- identification of indicator chemicals which are used to represent carcinogenic and non-carcinogenic risk at the site,

- exposure evaluation which includes the calculation of doses to potentially exposed populations,

- toxicological assessment of potential carcinogenicity of site indicator chemicals, non-carcinogenic effects and development of environmental standards, and

- characterization of the risks to a population caused by exposure to each indicator chemical.

This EA has been prepared subsequent to completion of RI and FS documents by the USEPA.

2.2 Indicator Chemicals

For the purpose of risk assessment, indicator chemicals are selected on a site-specific basis. The indicator chemicals must generally be prevalent to provide a representative analysis of risk for the site.

The selection and ranking of the indicator chemicals should follow the procedure outlined in the Superfund Public Health Evaluation Manual (USEPA, October 1986). A range and representative concentration for each chemical is calculated for each appropriate medium, as required by the procedure. As part of the indicator chemical selection process, toxicological information about each chemical is compiled using Appendix C of the Superfund Public Health Evaluation Manual (USEPA, October 1986). This information includes:

- toxicological class including potential carcinogens (PC) or non-carcinogens (NC),
- the severity-of-effect ratings value for non-carcinogens,
- the weight-of-evidence ratings for carcinogens, and
- toxicity constants for the various environmental media.

The chemicals identified at the specific site are subdivided into PCs and NCs. An indicator score (CT), which is the product of the chemical concentration and the toxicity constant (TC), is calculated for each medium and then summed to yield a total indicator score per chemical (IS). The chemicals are then ranked

numerically based upon decreasing indicator scores. The top-scoring compounds (based on IS values) are then re-evaluated based upon water solubility, vapor pressure, Henry's Law constant, and organic carbon partition coefficient (K_{OC}) to select the "most toxic, mobile and persistent chemicals at the site," according to the Superfund Public Health Evaluation Manual (USEPA, October, 1986).

2.3 Exposure Evaluation

The purpose of an exposure evaluation is to determine the intake of each indicator chemical by a potentially exposed population. After defining the modes of contaminant transport leading from the sources on the site to a point of exposure (Figure 2), the concentration of the indicator chemicals are determined in each medium with which a population may be exposed (i.e., exposure point concentration). A potentially exposed population is then defined and exposure doses are determined. Finally, the intake which results from the exposure is calculated.

The exposure evaluation (Figure 2) considers both the migration of contaminants from the site to potentially exposed populations and exposure from human use of the site by:

- evaluating fate and transport processes for the indicator chemicals,
- establishing exposure scenarios for each medium,
- determining exposures to potentially affected populations, and
- calculating doses and resultant intakes.

2.3.1 Evaluate Fate and Transport Process for the Indicator Chemicals

The first step in the analysis of exposure is to evaluate the fate and transport processes for the indicator chemicals in a qualitative manner, in order to consider the potential for releases from on-site and off-site sources of contamination in the exposure analysis. From this analysis any significant intermedia transport routes can be identified that may need to be evaluated in detail later in fate and transport modeling. Examples of the fate and transport processes of chemicals in the terrestrial, atmospheric, and aquatic environments are presented in Figures 3 and 4.

Examples of the environmental fates of the indicator chemicals include sorption onto soils and sediments, volatilization into the atmosphere, photochemical degradation, and bioaccumulation. Physical and chemical constants such as solubility and media partition coefficients are tabulated so that their importance in affecting fate and mobility of the contaminants can be evaluated.

2.3.2 Establish Exposure Scenarios for Each Medium

Exposure scenarios are determined by integrating information from the RI with knowledge about potentially exposed populations and their likely behavior. An exposure scenario is the qualitative connection between a source of a contaminant through one or a number of environmental media to a human population. The mode of exposure to the population such as inhalation, ingestion, or dermal contact is identified as part of the exposure scenarios.

2.3.3 Determine Exposures to Potentially Affected Populations

The next step is the quantitative determination of the exposure concentrations at the potential points of contact to the human populations. This step may be quite complicated since it requires knowledge of the contaminant source and its behavior in, and affect on, the environment between the site and any potentially exposed populations. The exposed populations for each medium may also be different. For example, this situation could arise if the direction of ground water flow was opposite to that of the predominant wind.

If the transporting medium can be treated as being at steady-state, monitoring data may be used to quantify exposure concentrations. If no data are available or if transient, increasing concentrations are suspected, models are used to predict concentrations, if possible. Many factors, including the fate processes mentioned previously, are considered when selecting the most appropriate model.

2.3.4 Calculate Doses to and Intakes by Potentially Exposed Populations

The resultant doses and intakes to potentially exposed populations are calculated once exposure concentrations in all media have been determined. A dose is defined as the amount of chemical contacting body boundaries (skin, lungs, or gastrointestinal tract), and intake is the amount of chemical absorbed by the body. To calculate dose and intake several factors must be considered, including the following:

- the amount of contaminated medium that contacts internal or external body surface during each exposure event,

- the amount of contaminant absorbed during each exposure event, and

- the frequency of each exposure event.

Doses and intakes are normally calculated together. For each exposure pathway under consideration, a dose per event is developed. This value quantifies the amount of contaminant contacted during each exposure event. "Event" may have different meanings depending on the nature of the scenario under consideration (e.g., each day's inhalation of contaminated air constitutes an inhalation exposure event). The quantity of contaminant absorbed during an event is calculated by considering the concentration of contaminant in the medium in which exposure occurs, the rate of contact with such medium (inhalation rate, ingestion rate, etc.), and the duration of each event.

Event-based dose values are converted to final intake values by multiplying the dose per event by the frequency of exposure events over the time frame being considered. Subchronic (short-term) exposure concentration is based on the number of exposure events that occur during the short-term time frame using maximum contaminant concentrations in the media to define dosage. It is intended to represent a 10 to 90 day exposure. Chronic (long-term) exposure concentration is based on the number of events that occur within an assumed 70-year lifetime using average contaminant concentrations in the media to define dosage.

Both doses and daily intakes are expressed in terms of mass of contaminant per unit of body mass per day by dividing daily exposures by an average body mass which is assumed to be 70 kg.

Both subchronic and chronic intakes are calculated. The Subchronic Daily Intake (SDI) is the projected human intake of a chemical averaged over a short time period, and is calculated by multiplying peak concentrations by human intake and body weight factors. It is used for subchronic risk characterization.

The Chronic Daily Intake (CDI) is the projected human intake of a chemical over a long time period, and is calculated by multiplying average concentrations by human intake and body weight factor. The CDI's are used for chronic risk characterization.

SDI's and CDI's are calculated by adjusting the short-term and long-term doses respectively to account for the amount of the doses absorbed by the body. Table 1 illustrates parameters used to calculate doses and intakes. Resultant intakes are then utilized in the risk characterization process. For carcinogens, the CDI values are used to assess carcinogenic risk and the SDI values are used to examine subchronic (acute) effects. For non-carcinogens, the intakes are used to evaluate acute and chronic effects.

Inhalation intakes are estimated based on the number of hours in each event, the inhalation rate of the exposed individual during the event, and the concentration of contaminant in the air breathed. One hundred percent of the contaminated mass inhaled is assumed to be absorbed. The formula for calculating event-based dosage is:

$$IEX = D \times I \times C$$

where:

IEX = estimated inhalation intake (mass of contaminant per event)
D = duration of an exposure event (hours per event)
I = average inhalation rate of exposed persons (cubic meters per hour)
C = contaminant air concentration throughout the exposure period (milligrams per cubic meter of contaminated air).

Subchronic (short-term) exposure resulting from inhalation is calculated using the maximum contaminant air concentration, while chronic (long-term) exposure is based on the average concentration. As a conservative approach USEPA recommends assuming that all of the inhaled contaminant is absorbed through the lungs.

Dermal intake is determined by the concentration of hazardous substance in a contaminated medium that is contacted, the body surface area contacted, and the duration of such contact. For exposure to contaminated water, dermal intake per event is calculated as follows:

$$DEX = D \times A \times C \times Flux$$

where:

DEX = estimated dermal intake per event (mass of contaminant per event)
D = duration of an exposure event (hours per event)
A = skin surface area available for contact (cm^2)
C = contaminant concentration in water (weight fraction)
Flux = flux rate of water across the skin (mass/cm^2/hr).

Subchronic intake resulting from each dermal exposure event is calculated using the maximum (short-term) contaminant concentrations in water. Chronic intake is based on average (long-term) contaminant concentrations.

Intake resulting from ingestion of water-borne contaminants is determined by multiplying the concentration of the contaminant in the water by the amount of water ingested per day and the degree of absorption.

2.4 Toxicity Assessment

A toxicity assessment (Figure 5) of the selected indicator chemicals is conducted to identify potential applicable and relevant standards and to develop a data base against which exposure point intakes can be compared during the risk characterization evaluation. The evaluation includes consideration of experimental studies using mammals and aquatic nonmammalian species (where available), as well as relevant standards for humans.

The evaluation presents summaries of health effects data, pharmacokinetics and metabolism, toxic and carcinogenic effects, and applicable and relevant standards available for the indicator chemicals. Because of its major impact on the risk evaluation, the procedures used for classifying animal and human carcinogens by the USEPA, as well as by the International Agency of Research on Cancer of the World Health Organization (IARC), and the attendant uncertainties, are presented below.

Evaluation of carcinogenicity involves two steps: (1) the identification of potential carcinogens, and (2) the quantitative determination of carcinogenic potency.

2.4.1 Identification of Carcinogens

Evidence of possible carcinogenicity in humans comes primarily from long-term animal tests and epidemiological investigations. Results from these studies are supplemented with information from short-term tests, pharmacokinetics studies, comparative metabolism studies, structural-activity relationships, and other relevant information sources.

When judging qualitative evidence of carcinogenicity, USEPA as well as IARC have adopted a policy of "weight-of-evidence", meaning that the quality and adequacy of all relevant data on responses induced by a possible carcinogen using different procedures will be considered. There are three major steps in determining the weight-of-evidence for carcinogenicity:

- characterization of the evidence from human studies and from animal studies individually,

- combination of the two types of studies into a final indication of overall weight-of-evidence for human carcinogenicity, and

- evaluation of all supportive information to determine if the overall weight-of-evidence should be modified.

Further details concerning the classification system of USEPA and use of this data in the risk assessment process are presented in Appendix A.

2.4.2 Determination of Carcinogenic Potency

The second phase in carcinogen assessment involves the quantification of risk. Experimental studies of carcinogenic

effects utilizing the low exposure levels usually encountered in the environment usually are not feasible. Therefore, various mathematical models have to be used for extrapolation from the high doses used in animal bioassays down to the dosages of interest in connection with exposure to ambient environmental concentrations. However, since the resolution power of animal studies is not adequate for precise elaboration of the dose-response curve, extrapolating from a high dose to a low dose introduces a level of uncertainty which may amount to orders of magnitude. Given the recognized differences in carcinogenic response between species and between strains of the same species, it is clear that additional uncertainties will be introduced when quantitative extrapolations (e.g., between rodents and humans) are made. Among various proposed models of quantitative extrapolation, USEPA recommends the use of a linearized multistage model "unless there is evidence on carcinogenesis mechanisms or other biological evidence that indicates the greater suitability of an alternative extrapolation model, or there is statistical or biological evidence that excludes the use of a linearized multistage model" (Federal Register, 1984).

The carcinogenic potency of a chemical is often expressed in terms of a potency factor which is the upper 95 percent confidence limit on the probability of response per unit intake (mg/kg, etc.) of a chemical over a lifetime. USEPA's Carcinogen Assessment Group (CAG) has evaluated more than 54 chemicals as suspect human carcinogens and developed relative carcinogenic potency factors for each chemical.

The toxicity information presented herein relies primarily on information provided in the Superfund Public Health Manual (USEPA, October 1986).

2.5 Risk Characterization

As shown in Figure 6, the risks to potentially exposed population from exposure and subsequent intakes of the indicator chemicals are determined through the consideration of:

- comparison with environmental standards,
- non-carcinogenic risk, and
- carcinogenic risk.

2.5.1 Comparison with Environmental Standards

Exposure point concentrations of the indicator chemicals are compared to potentially applicable or relevant and appropriate standards as defined by the National Contingency Plan (NCP) and identified in the CERCLA compliance policy memo which is an appendix to the NCP, as well as additional requirements identified in the CERCLA reauthorization statute (SARA). At the present time, USEPA considers drinking water maximum contaminant levels (MCLs) and maximum contaminant level goals (MCLGs), national ambient air quality standards (NAAQS), federal ambient water quality criteria, and federally-approved state water quality standards developed under the Clean Water Act to be potentially applicable, or relevant and appropriate ambient concentration requirements.

2.5.2 Non-carcinogenic Risk

The Hazard Index method is used for assessing the overall potential for non-carcinogenic effects posed by multiple chemicals. This approach assumes that multiple subthreshold exposures could result in an adverse effect and that the

magnitude of the adverse effect will be proportional to the sum of the ratios of the subthreshold exposures to acceptable exposures. This can be expressed as:

$$\text{Hazard Index} = E_1/AL_1 + E_2/AL_2 + \ldots + E_i/AL_i$$

where:

E_1 = exposure level (or intake) for the i^{th} contaminant

AL_i = acceptable level (or intake) for the i^{th} contaminant

For a single contaminant, there may be a potential adverse health effect when the hazard index exceeds unity. For multiple chemical exposures, hazard indices, if summed, may result in an overall hazard index that exceeds one, even if no single chemical exceeds its acceptable level. However, the assumption of additivity should only be made for compounds that produce the same toxic effect by the same mechanisms of action.

USEPA has developed some preliminary information regarding Acceptable Intakes for Subchronic Exposures (AISs) and Acceptable Intakes for Chronic Exposures (AICs) (Mabey, W.R., et al, 1982). Where these are available, they are used as acceptable levels for subchronic and chronic exposures, respectively.

2.5.3 Carcinogenic Risk

For potential carcinogens, risks are estimated as probabilities. The carcinogenic potency factor, which is the upper 95% confidence limit of the probability of a carcinogenic response per unit intake over a lifetime of exposure, converts estimated Chronic Daily Intakes (CDI's) directly to incremental risk values. In general, because only relatively low CDI's are likely to result from environmental exposures, the USEPA methodology

assumes that the exposure will be in the linear portion of the dose-response curve. Based on this assumption, the slope of the dose-response curve is equivalent to the carcinogenic potency factor, and the risk is directly related to the CDI at low levels of exposure. The low-dose carcinogenic risk equation is:

$$\text{Risk} = \text{CDI} \times \text{Carcinogenic potency factor}$$

Once an estimate of risk has been obtained, the question arises as to what level of risk is acceptable. The USEPA Office of Emergency and Remedial Response (OERR) is developing a strategy which will articulate the general framework in which they suggest ground water cleanup decisions be made. In this framework, it is advised that at least one remedial alternative be developed at a site which would attain applicable and appropriate health standards for carcinogens where they are available, or a 10^{-6} risk level for carcinogens without standards for current and potential exposure. Such an alternative should be designed to attain these levels within a short period of time and should be used as a point of departure in analyzing a range of alternatives. A target range for all alternatives should be the 10^{-7} to 10^{-4} risk level range.

3.0 INDICATOR CHEMICALS

3.1 Selection of Indicator Chemicals

Because indicator chemicals have already been established for ECC as part of the USEPA EA development process, no attempt has been made in this evaluation to rederive, only evaluate, this list of compounds.

As detailed in the February, 1987 PRP comment report on the USEPA RI/FS and Combined Alternatives Analysis, methylene chloride is

not an appropriate indicator chemical for the ECC site. On page 4-68 of the ECC RI report the USEPA states:

> "Methylene chloride appeared in all samples including the blank and may be a sample bottle contaminant ... As a result it is not believed that ECC is the source of this potential contamination."

The presence of methylene chloride does not indicate a risk which requires mitigation. Therefore, methylene chloride has not been included in the list of indicator chemicals for this EA.

3.2 Final List of Indicator Chemicals

The list of indicator chemicals for the ECC site is as follows (from Table 5-1, EPA ECC-RI):

- Chloroform
- 1,1,2-Trichloroethane (1,1,2 TCA)
- 1,1,1-Trichloroethane (1,1,1 TCA)
- Trichloroethene (TCE)
- Tetrachloroethene (PCE)
- Ethylbenzene
- Toluene
- Phenol
- PCBs
- Bis(2-ethylhexyl)phthalate
- Di-n-butyl phthalate
- Diethyl phthalate
- Dimethyl phthalate

4.0 TOXICITY ASSESSMENT OF THE INDICATOR CHEMICALS

A brief summary of physical and chemical properties of the indicator chemicals is presented in Table 2. A full discussion of health effects of the indicator chemicals (with references) is provided in Appendix D. Table 3 summarizes toxicological information for the indicator chemicals, including the environmental standards, acceptable daily intakes for non-carcinogenic effects, and potency factors for potential carcinogens.

5.0 TOXICITY EVALUATION PROCESS

The toxicity evaluation of the selected indicator chemicals for the ECC site follows the procedure outlined in Section 2.4. The process involves three components:

- comparison with environmental standards,
- evaluation of non-carcinogenic risk
- assignation of carcinogenicity

5.1 Comparison with Environmental Standards

As discussed in Section 2.5, evaluation of exposure point concentrations compared with environmental standards are essential to understanding site-related levels of environmental risk and damage. Potentially applicable, or relevant and appropriate requirements for each indicator chemical are presented in Table 3.

5.2 Non-carcinogenic Effects

Non-carcinogenic risks are evaluated primarily by comparing site-related doses to acceptable daily intakes, established to protect

against various types of acute and chronic effects. Acceptable daily intakes are included in Table 3.

5.3 Assignment of Carcinogenicity

The decision to classify a compound as a potential carcinogen has serious consequences for the conduct of quantitative risk assessments. Wrongly attributing a compound's carcinogenicity can result in severe over- or under-estimation of carcinogenic risk. Carcinogenic risk at CERCLA sites is normally the most restrictive component of the EA process; therefore, the appropriateness of cleanup decisions quite likely depends upon the accuracy of the determination of carcinogenic risks.

The level of evidence for carcinogenicity for the indicator compounds is discussed in detail in Appendix A. A brief summary of that discussion is given below.

There is a significant controversy in the international scientific community surrounding the classification of trichloroethene (TCE). USEPA has classified TCE as a potential (Class B2) carcinogen. However, USEPA's interpretation of mouse liver tumors observed in long-term studies and the appropriateness of the use of the linearized multistage model for calculation of carcinogenic potency have not been widely accepted by the scientific community. IARC has determined that there is insufficient evidence to classify TCE in regard to carcinogenicity at this time (IARC, 1979). In this EA, USEPA's classification of TCE has been accepted and therefore, TCE has been included in the carcinogenic risk assessment.

Chloroform and PCBs are considered as probable human carcinogens by both USEPA and IARC. Tetrachloroethene is considered a probable carcinogen by USEPA, a possible carcinogen by IARC. Bis(2-ethylhexyl)phthalate is considered to be a possible human

carcinogen by USEPA, but is not ranked by IARC because of the lack of evidence in human studies (IARC, 1979 and 1982). As with TCE, USEPA's classification has been accepted in this EA.

Both USEPA and IARC consider 1,1,1-trichloroethane to display limited evidence of human carcinogenicity.

6.0 EXPOSURE ASSESSMENT

This section evaluates potential exposure of human populations to contaminants associated with the ECC site. The exposure assessment process leads to the determination of intakes of each indicator chemical by each potentially exposed population through the following steps:

- evaluation of the sources of contamination, and analysis of the applicable fate and transport processes for the indicator chemicals,

- establishment of exposure scenarios for each medium,

- determination of exposures to potentially affected populations, and

- calculation of doses and resultant intakes.

6.1 Sources of Contamination and Evaluation of Fate and Transport Processes for the Indicator Chemicals

The original and current sources of contamination at the ECC site are discussed in detail in Chapters 1 through 4 of the ECC RI report. The major source of continuing contamination is residually contaminated soils. Initial contamination of these

soils occurred as a result of previous spillage and leakage of drummed waste, as well as potential on-site cooling pond leakage. Although accurate mass balance calculations of expected remaining contamination are not possible, the magnitude of the remaining contamination can be inferred from sampling results. For purposes of this assessment, the contaminated soils remaining on-site are identified as the current source of contamination.

The relevant physical and chemical properties of the indicator chemicals are presented in Table 2, and the processes influencing the fate of the indicator chemicals are evaluated in Table 4. Detailed discussions of relevant fate and transport mechanisms for each of the indicator chemicals are included in Appendix C.

From the contaminated soils, three modes of direct environmental transport of contaminants are possible considering the present condition of the site: (1) leaching of contaminants from on-site soils and subsequent transport in ground water, (2) volatilization of contaminants, and (3) transport via the food chain.

6.2 Potential Exposure Scenarios

Potential exposure scenarios considered for the ECC site are listed in Table 5. These scenarios consist of the connections between the sources of contamination, the possible transport media for contaminants, the resulting exposure points where human contact with contaminants is possible, and the potential routes of exposure. Scenarios are also evaluated as applicable or non-applicable to this assessment. We have identified the following exposures for further quantitative analysis (USEPA Draft Superfund Exposure Assessment Manual, 1986):

Air

- Volatilization of contaminants from on-site soils.

Surface Water

- Dermal exposure to contaminants in surface water during recreational or play activities.

- Bioaccumulation and transport via the food chain in Finley Creek fish.

Soil

- Dermal exposure to contaminants in soil during play activities, both on and off-site, with incidental ingestion of 0.1 grams of soil. Incidental dust inhalation is included in these calculations.

- Ingestion of contaminants in soil during pica behavior, both on and off-site.

The following exposures were not evaluated further for the reasons given:

Ground Water

- Dermal exposure or ingestion of shallow saturated zone ground water, since this zone is not suitable for development of a long-term water supply due to limited yield.

Air

- Inhalation of fugitive dusts from the site, as future use or non-use will result in either impermeable capping or vegetative covering. Also, the scope of future soil disturbance, if any, cannot be predicted. Incidental dust inhalation is included as part of the soil ingestion calculations.

- Volatilization from surface waters, as this impact is insignificant compared to volatilization from on-site soils in this case.

Surface Water

- Ingestion of contaminants found in surface water bodies, since neither Finley Creek nor Unnamed Ditch is a drinking water source.

- Dermal exposure to soils contaminated from overland flow, as such flow is directed to the streams and, in effect, does not impact other, non-site areas.

6.3 Determination of Exposures to Potentially Affected Populations

The exposure scenarios identified for further analysis require the quantitative determination of contaminant concentrations at the following exposure points:

- Concentration of volatilized contaminants in ambient air on the site,

- Concentration of contamination in surface water in the Unnamed Ditch and Finley Creek,

- Concentration of contaminants in on-site soils.

Land use and demography of the surrounding area are discussed in the RI. The only sensitive sub-population identified within a three mile area of the site are children; therefore, they are assessed separately in the EA.

Only unqualified data were used in calculating exposure concentrations. Average concentrations were calculated using data generated from samples taken across the entire site.

A detailed discussion of techniques used for determination of both short and long term concentrations at each of the exposure points follows.

6.3.1 Contaminants in Air On-Site

Volatilized contaminant releases having the potential to be present on-site may originate through the present soil cover. The rate of emission at the soil surface of volatile organics originating from contaminated ground water was estimated using the following method presented in the Draft Superfund Exposure Assessment Manual (USEPA 1986a):

$$E_i = D_i C_{Si} \quad A \; P_t^{\,4/3} \; \frac{M_i}{d_{sc}}$$

where:

E_i = emission rate of component i (g/sec)
D_i = diffusion coefficient of component i (cm^2/sec)
C_{Si} = saturation vapor concentrations in component i, (g/cm^3)
A = exposed area (cm^2)
P_t = total soil porosity (assumed to be 0.35)
d_{sc} = effective depth of soil cover (cm)
M_i = weight fraction of toxic component i in the waste (g/g)

The simplified equation used to compute downwind concentrations from a point source is:

$$C(x) = \frac{Q}{Pi\ S_y\ S_z\ u}$$

where:

C(x) = concentration of substance at distance x from the release point (g/m^3)
Q = emission rate of the substance from the release point (g/sec)
Pi = 3.14159
S_y = dispersion coefficient in the lateral direction (1/m);
S_z = dispersion coefficient in the vertical direction (1/m)
u = mean wind speed (m/sec)

Values of S_y and S_z were obtained from the relevant graphs presented in the Draft Superfund Exposure Assessment Manual (USEPA, 1986a). To obtain a conservative subchronic estimate, stability class F and wind speed of 1 m/s directed toward the

receptor point were assumed. To obtain long-term, chronic concentrations, D stability and a mean wind speed of 3 m/s directed toward the receptor point were assumed. All exposure concentrations were modeled using maximum and average emission rates, (for subchronic and chronic concentrations respectively), which were based on the maximum and average concentrations of contaminants found in the soil. It was assumed that the wind blows towards the exposure point 100% of the time. The resulting short-and long-term (subchronic and chronic) exposure point concentrations are given in Table 6.

6.3.2 Concentrations in On-Site Soils

Short and long-term concentrations were derived from the results of the Remedial Investigation sampling, and are based on maximum and mean concentrations reported in shallow soils (Tables 4-1 and 4-6, ECC-RI). These values are listed in Table 6.

For volatile organics, the combined decay of the mass of source contaminants in the soil was calculated based upon volatilization and hydrolysis phenomena contributing to that decay.

Volatilization. Based upon the volatile emissions equation presented above in the air contaminants discussion taken from the draft Superfund Exposure Assessment Manual (USEPA, 1986a), volatilization was determined to be a first order reaction. The decay rate constant was determined to be the following:

$$k_i^v = E_i / M_i p \, Ad_c, \ sec^{-1}$$

where:

p = denisty of soil (assumed to be 2.0 g/cm^3)

d_c = depth of contaminated zone (assumed to be 100 cm)

Hydrolysis. Hydrolysis half-lives for various compounds are presented in "Water-Related Environmental Fate of 129 Priority Pollutants" (USEPA, 1979). Generally, hydrolysis half-lives for volatile organics are less than a year. Although volatilization is typically considered a more significant fate process than hydrolysis for these volatile organics, extended travel times in the ECC case make hydrolysis a fate which will significantly impact residual concentrations.

The question arises as to whether hydrolysis rates in saturated soils might be more or less than that in water. According to Valentine (1986), sorption could lead to increased, decreased, or unchanged hydrolysis rates. Burkhard and Guth (1979) found that the rate of hydrolysis in soils was increased compared to that in pure aqueous solution, but that rates decreased with the extent of sorption. In contrast to these results, Konrad and Chester (1969) observed that the first order rate constants were directly related to sorption. Given the likely hydrolysis of volatile organics in the soil at ECC, but with the uncertainty of precise predictability in the respective hydrolysis rates, a uniform hydrolysis half-life of two years was used for decay determination in this case. Therefore, the first order rate constant to express this decay is as follows:

$$k_i^h = -\ln 0.5 \; / \; t^{1/2}$$

No hydrolysis was assumed for ethylbenzene or toluene.

Leaching. Concentration of leachate entering the ground water table from the source was calculated using the following equation:

$$C_L = (K)(C^x)(S^y)$$

where:

C_L = Concentration of contamination in leachate
K = 0.044
x = 0.71
y = 0.31

C = concentration of contaminant, mg/kg
S = solubility, mg/l

The above USEPA GLM model was taken from the November 27, 1985 Federal Register.

Net recharge through the source mass was determined to be 4 inches per year based upon information provided by the Indiana Geological Survey. Using that recharge, a check was made to confirm that a significant portion of the mass is not lost each year which would impact the volatilization and hydrolysis decay calculations. The combined volatilization and hydrolysis decay as calculated provides a realistic and conservative approach since leaching decay is ignored in calculating residual mass concentration.

Combined Decay of Mass. Combining the above, then, the model used to determine resulting leachate concentration from initial soil concentrations $[(C_L{}^t{}_i]$ is as follows:

$$(C_L{}^t)_i = K[(C_0)_i \exp[-(k_i{}^T)_i t]_i{}^{0.71}(S)_i{}^{0.31}]$$

$$\text{where: } k_i{}^T = k_i{}^v + k_i{}^h$$

Similarly, the concentration of source material remaining at any time (C^t_i) will equal:

$$C^t_i = C_o \exp[-(k_i^T)_i t$$

where: $k_i^T = k^v + k^h$

6.3.3 Concentrations in Surface Water

The following presents methodology used to calculate residual contaminant concentrations into Unnamed Ditch. Contaminant was assumed to enter the satuated glacial till via leachate as calculated above in Section 6.3.2. A saturated thickness of four meters was used, and it was assumed that leachate spread instantly across the four meters at the start of its travel from the source to Unnamed Ditch. A distance of 100 feet from the source to the Unnamed Ditch was used to predict travel times.

Retardation due to sorption of individual chemicals in relation to ground water flow was calculated using the following methodology:

1. Water-organic carbon partition coefficients (Log K_{OC}) were calculated based upon the following formulae taken from the Handbook of Chemical Property Estimation Methods (Lyman and others, 1981).

   a. For halogenated hydrocarbons:

$$\text{Log } K_{OC} = -0.557 \log S + 4.277$$

S = solubility in micromoles per liter

b. For aromatic hydrocarbons:

$$\text{Log } K_{OC} = -0.54 \log S + 0.44$$

S = solubility in mole fraction

c. For phthalates and PCBs:

$$\text{Log } K_{OC} = -0.55 \log S + 3.64$$

S = solubility in milligrams per liter

2. The soil-water partition coefficients (K_p) were calculated based upon methodology presented in "Remote Detection and Preliminary Hazard Evaluation of Volatile Organic Contaminants in Ground Water" by Marrin (1984):

$$K_p = (\text{sand fraction}) \times 0.2 \times OC + K_{OC} \times (\text{fines fraction}) \times OC \times K_{OC}$$

where: OC = organic carbon content = 0.002

Sands fraction = 0.40

Fines fraction = 0.60

3. The retardation factor (R), i.e., the relative velocity of the chemical in relation to the velocity of water, was calculated using Marrin (1984) as follows:

$$R = 1 + (K_p \times p \times 1/VW)$$

where: p = density of soil (assumed to be 2.0 g/cm^3)

VW = volumetric water content of media (assumed to 0.1)

Ground water velocity was determined using Darcy's Law. Resulting travel times for the various indicator chemicals to travel 100 feet to Unnamed Ditch are presented in Table B-1. Using a methodology similar to that presented above for decay of the contaminant source area, volatilization and hydrolysis (volatiles only) during ground water transport was applied to reflect continuing decay of contaminant concentrations in the leachate. In this case, the relative concentration of a contaminant chemical just prior to entering Unnamed Ditch $[(C_r{}^t)_i]$ in relation to its concentration as it first enters the ground water table 100 feet away $[(C_{gw}{}^t)_i]$ can be expressed as follows:

$$(C_r{}^t)_i/(C_{gw}{}^t)_i = \exp[- (K_i{}^T)_i{}^{gw}\ t_i]$$

where: $(K_i{}^T)_i{}^{gw} = g_i + (K_i{}^h)_i$

g_i = $2/3\ D_i\ d/DH$

d_i = the gas - liquid partition coefficient for each chemical.

H = depth of soil cover

D = depth of saturated zone

Discharge from the saturated till to the Unnamed Ditch was estimated using Darcy's Law. Due to a lack of data, the flow gradient to the Unnamed Ditch in the till was assumed equal to the average ground surface gradient. In addition, it was conservatively assumed that the entire thickness of till discharges to the Unnamed Ditch. The flux (Q) from the saturated till to the Unnamed Ditch was calculated as follows:

$$Q = KAi$$

where:

K = hydraulic conductivity = 8.64×10^{-3} m/day
A = cross-sectional area of discharge = (200m)(5.5m)=1100m^2
i = gradient of flow = 0.04 m/m

therefore:

Q = (8.64×10^{-3}) (1100) (0.04) m^3/day
= 0.38 m^3/day (13.41 ft^3/day)

Flow in the Unnamed Ditch was measured by the USEPA to be 244.84 m^3/day (0.1 ft^3/sec) along the ECC site. Therefore discharge concentration $(C_r{}^t)_i$ from the saturated till to the surface water of the Unnamed Ditch is diluted by a factor of 0.0016.

6.3.4 Fate of Non-volatile Organics

Non-volatile indicator organic contaminants were handled in the following ways for purposes of fate and exposure analysis:

1. Phthalates. Phthalates were assumed not to decay by either volatilization nor hydrolysis. Phthalates are, however, generally biodegradable (Lyman, et. al., 1981; USEPA, 1979), and because of high sorption characteristics (USEPA, 1979) and resulting very long travel times between the source and Unnamed Ditch, it is assumed that phthalates will be reduced to insignificant levels through biodegradation before reaching the ditch. No attempt was made to predict biodegradation rates, as appropriate decay

constants can only be accurately determined through treatability evaluation on actual contaminant samples.

2. Phenol. No volatilization or hydrolysis was assumed for phenol. However, phenol is readily biodegradable (Lyman, et. al., 1981; USEPA, 1979). It is assumed that it would be reduced through biodegradation to insignificant levels prior to reaching Unnamed Ditch.

3. Arochlor 1260. Although volatilizing at a relatively slow rate, high sorption tendency (USEPA, 1979) and resulting extended travel time to Unnamed Ditch (11,111 years) indicate some decay due to volatilization is probable prior to PCBs reaching the ditch. No hydrolysis or biodegradation of PCBs was assumed.

6.4 Calculation of Doses and Intakes

Routes of exposure used in this investigation for the calculation of intakes are summarized in Table 7. The parameters of exposure assumed for description of the subchronic and chronic exposure scenarios are given in Table 8. It should be noted here that behavior over a 24 hour exposure scenario must be realistic; that is, not more than a total of 24 hours per day of exposure from all scenarios is possible.

Subchronic and chronic exposures for all potentially exposed populations are presented in Tables 9 thru 14.

It is also important to note that all values shown in the EA tables as 0.00E+00 signify that calculations were completed, but that they resulted in values less than 1×10^{-10}.

7.0 Endangerment Assessment

7.1 Comparison to Applicable and Appropriate or Relevant Standards

Comparison of existing indicator chemical concentrations at all exposure points with potentially applicable and appropriate or relevant standards (ARARs) as defined by the U.S. USEPA are presented in tabular form in Table 15.

7.2 Calculation of Short-Term (subchronic) Exposure Hazard

Subchronic intakes were used to assess the short-term exposure effects, for both noncarcinogenic and carcinogenic compounds. Total subchronic hazard for each potentially exposed population is presented in Table 16. The dermal and ingestion intakes were compared to the oral acceptable intake. Similarly, the inhalation intake was compared to the inhalation acceptable intake. Total subchronic hazard for all population does not exceed the recommended safe hazard index value of one.

7.3 Calculation of Long-Term (chronic) Exposure Hazard

7.3.1 Noncarcinogenic Hazard

Chronic intakes were used to assess the long-term exposure hazard of noncarcinogens. Total noncarcinogenic (chronic) hazard for each potentially exposed population is presented in Table 17. The oral acceptable intakes were used to assess the dermal and ingestion noncarcinogenic chronic hazards of the indicator

compounds, while the inhalation acceptable intakes were used to assess the inhalation chronic hazard. The total noncarcinogenic hazard index (NHI) does not exceed the recommended safe hazard index value of one for on- or off-site populations.

7.3.2 Carcinogenic Risk

Chronic intakes were also used to assess carcinogenic risk. Total carcinogenic risk is presented in Table 18, as well as potency factors for each indicator compound. The total "weighted" lifetime carcinogenic risk is below the maximum acceptable USEPA level of 10^{-4} and within the 10^{-7} to 10^{-4} generally accepted range for Superfund sites.

The highest risk group at 2×10^{-4} are "pica" behavior children; the highest risk for non "pica" behavior is 2×10^{-5}.

7.4 Special Cases for Ethylbenzene, Toluene and PCBs

For the combined Risk Assessment determination presented above, it has been assumed that concentrations of ethylbenzene, toluene and PCB's reaching Unnamed Ditch are zero. Because of extremely long travel times predicted for these chemicals to reach Unnamed Ditch, the concentrations of these compounds reaching the ditch during the period of assumed combined exposure (70 years) will, in fact, be zero. It is only in later years, when these compounds are predicted to reach the ditch, that they will present exposure risks. In an effort to predict that future risk, both subchronic and chronic exposure were calculated for each of the three chemicals independently. Subchronic hazard indices were calculated to be less than 3×10^{-8} and 6×10^{-10} for toluene and ethylbenzene, repectively. Likewise, chronic hazard indices were calculated to be less than 2×10^{-8} and 3×10^{-9} for these same chemicals, respectively. Chronic carcinogenic risk for PCB's was calculated to the less than 7 x

10^{-11}. The calculations for PCBs included both dermal exposure as well as bioaccumulation in fish caught from Finley Creek.

7.5 Prediction of Future Extent of Contamination

Based upon information presented in the ECC RI related to the physical definition of the ECC site and immediate surroundings, in addition to fate and transport of contaminants predicted in this EA, there is no reason to suspect that significant contamination will move off-site over time. There is expected to be contaminant migration through shallow saturated glacial till to the east and southeast at rates predicted. Because of decay of contaminant mass in the source area, as well as decay of leachate from that mass as it moves towards the Unnamed Ditch, extremely small concentrations are expected in the Unnamed Ditch in the future years, as can be seen in Appendix B. Similarly, predicted off-site movement of volatile organics through the air will have de minimus and decreasing health effects.

7.6 Endangerment Summary

This analysis has shown that endangerment risk from the contaminants at the ECC site are within the range normally found acceptable to USEPA at Superfund sites. It is important to note that these risks were calculated using the most conservative assumptions which, when considered together, suggest that the probability of the kinds and magnitude of exposure predicted are highly unlikely and that a more realistic level would be significantly lower than those values shown.

The analysis shows that the largest portion of risk calculated is due to presumed dermal absorption and ingestion of on-site soils. Little or no risk due to off-site migration and exposure was found. These conclusions suggest that simple access restriction to the site would substantially reduce exposure risk to below

those levels determined herein. In summary, if access restrictions are implemented, endangerment from the ECC site is effectively eliminated.

FIGURES

FIGURE 1. THE RISK ASSESSMENT PROCESS.

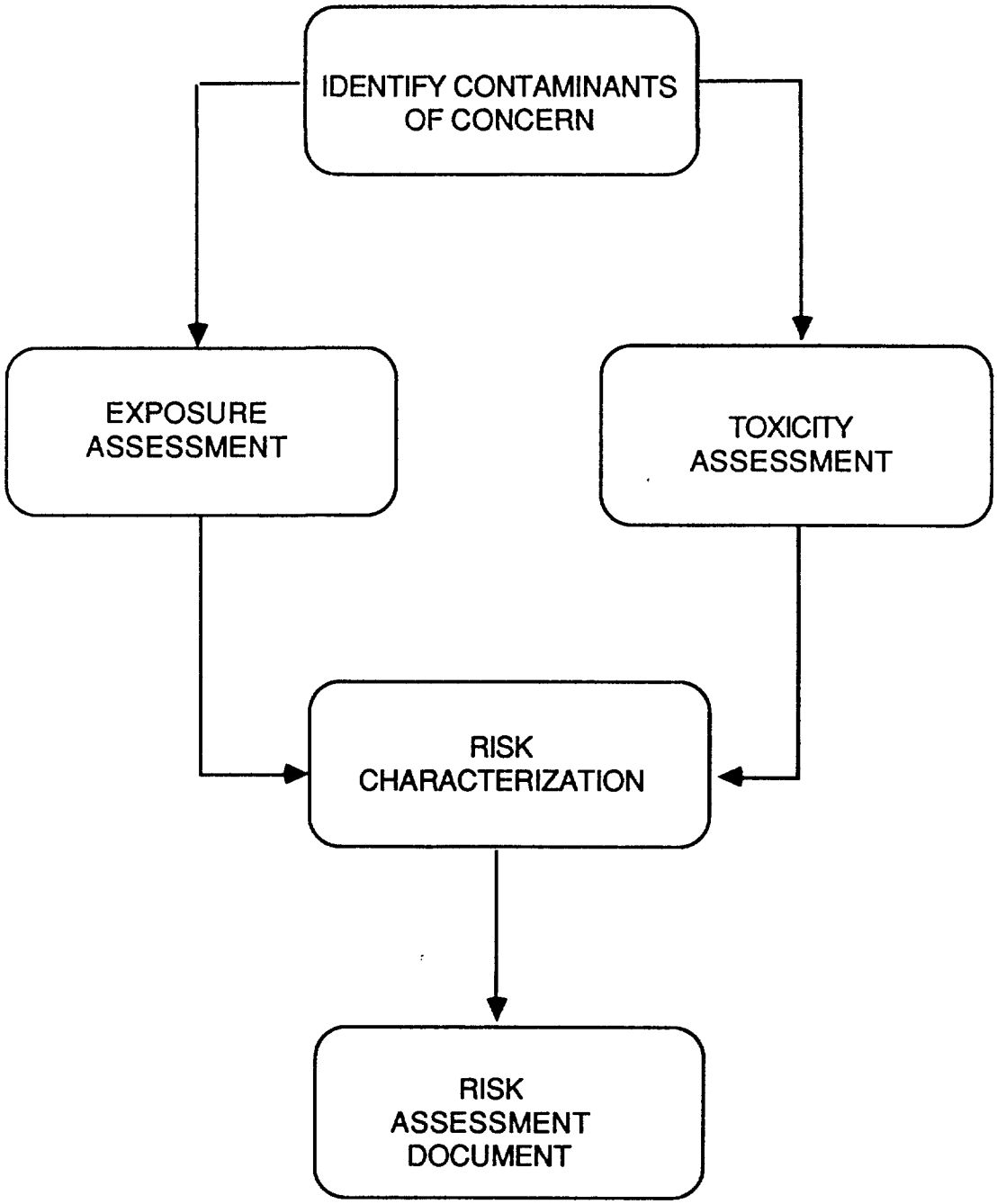


FIGURE 2. THE EXPOSURE ASSESSMENT PROCESS

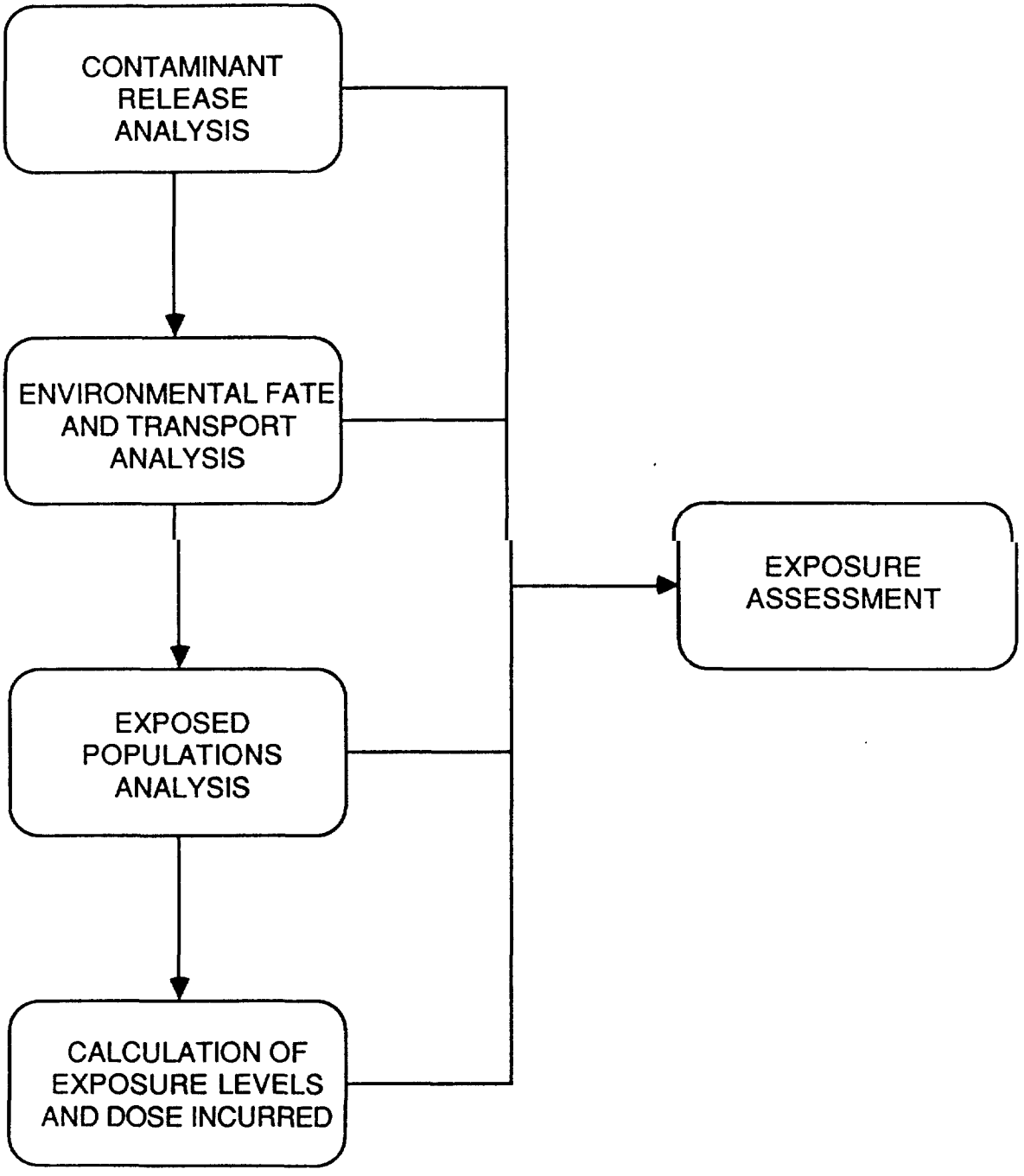


Figure 3
Fate and Transport Processes of Chemicals in the Aquatic Environment

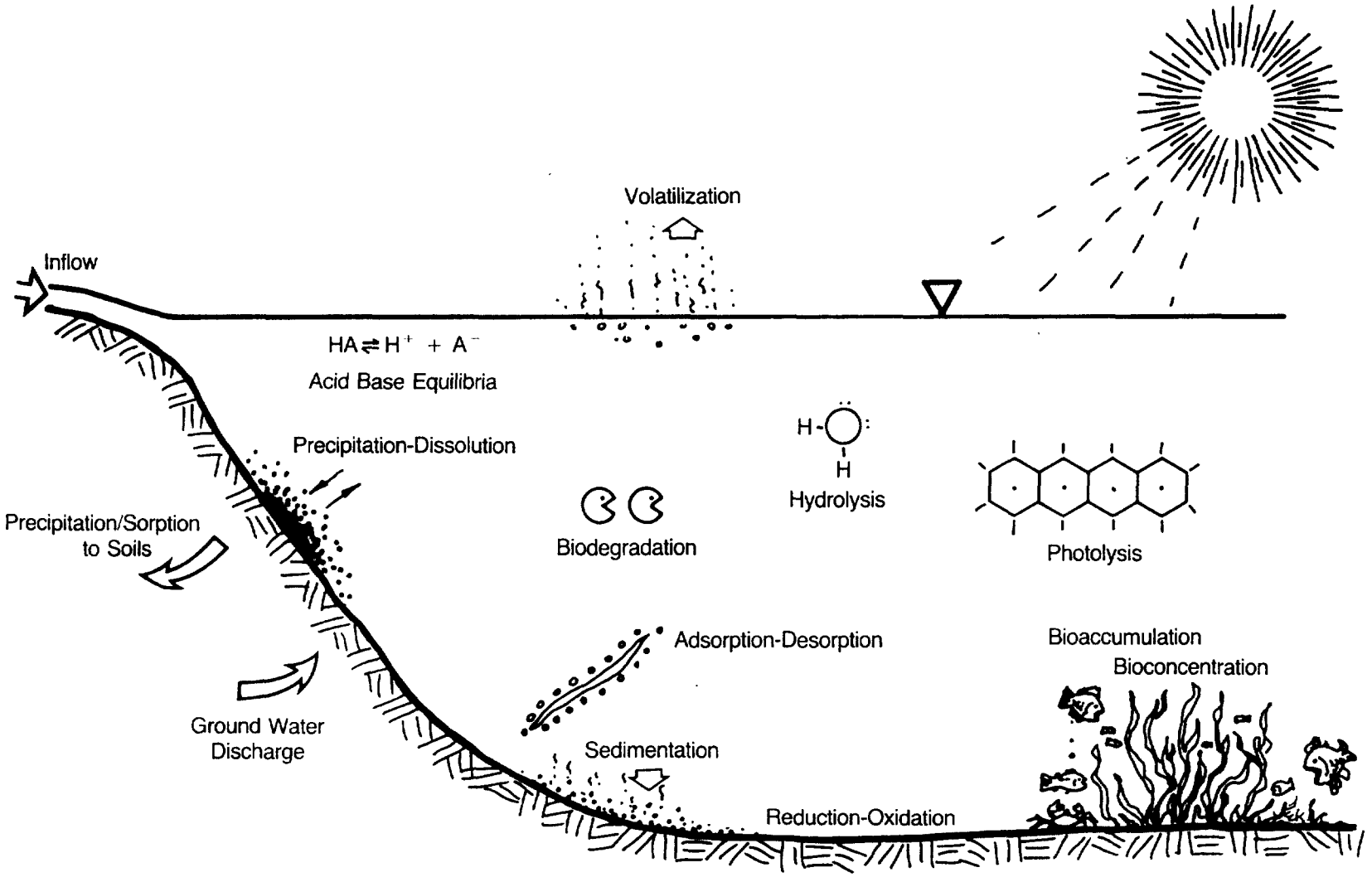


Figure 4
Fate and Transport Processes of Chemicals in the Terrestrial and Atmospheric Environment

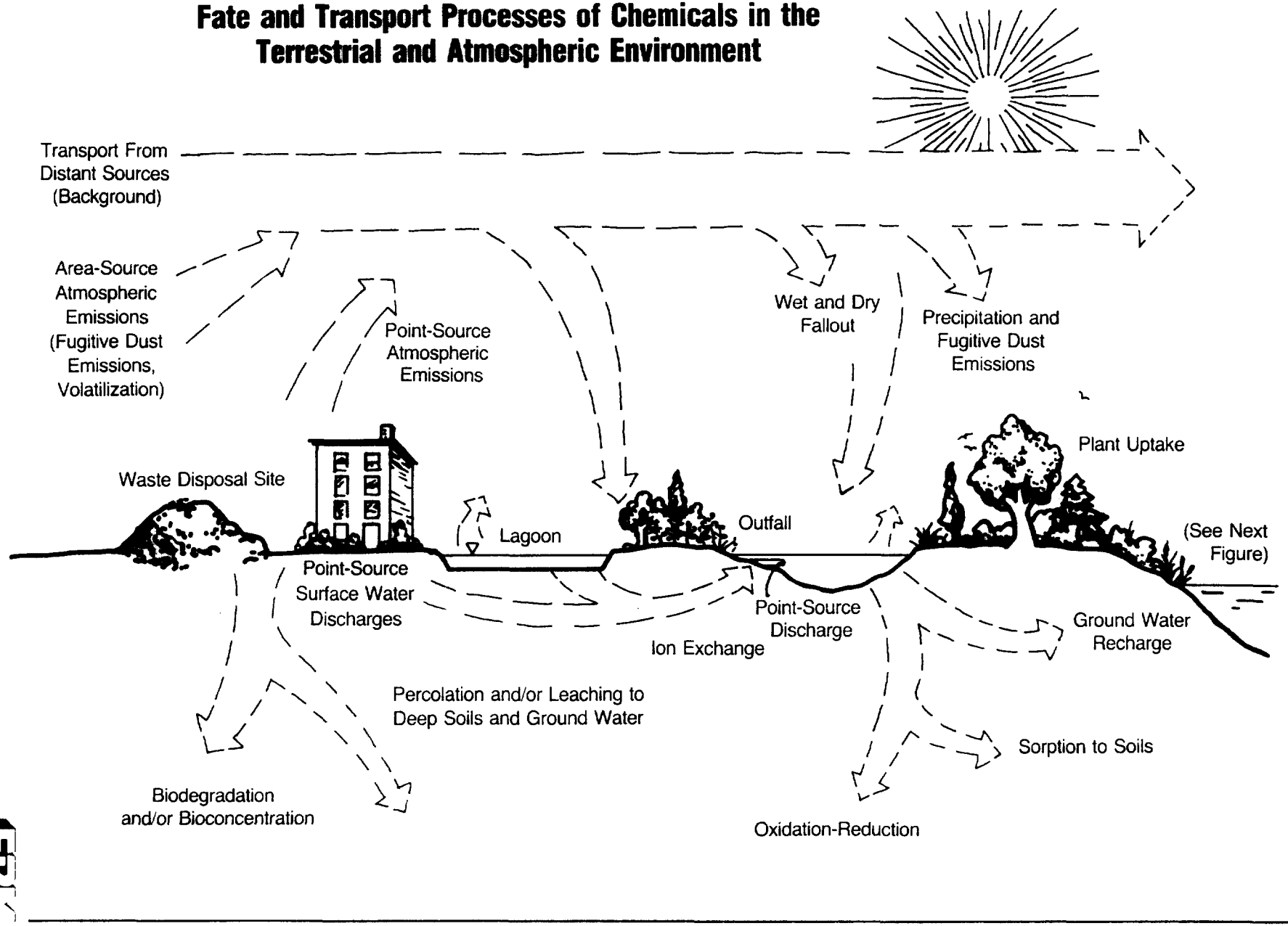


FIGURE 5. THE TOXICITY ASSESSMENT PROCESS

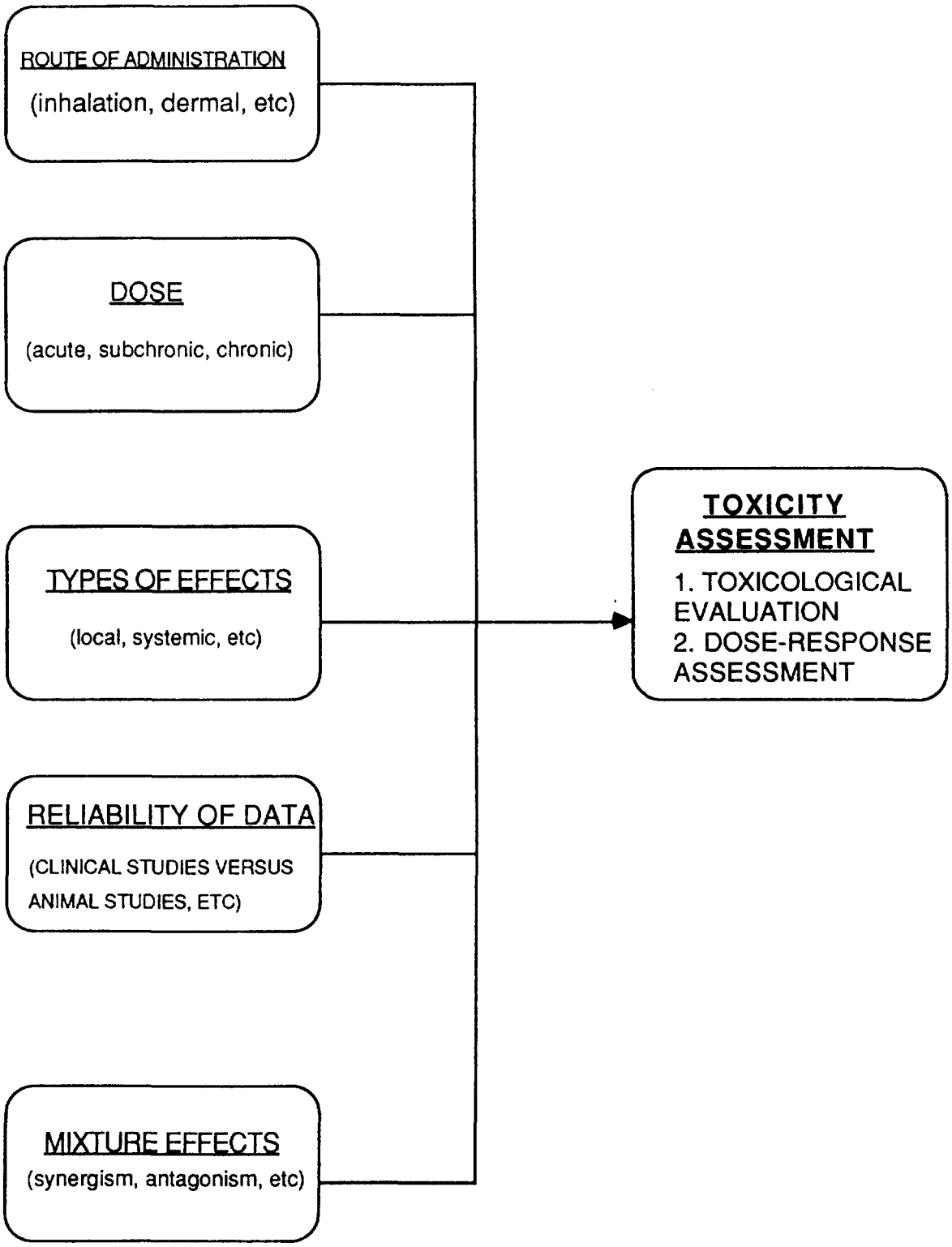
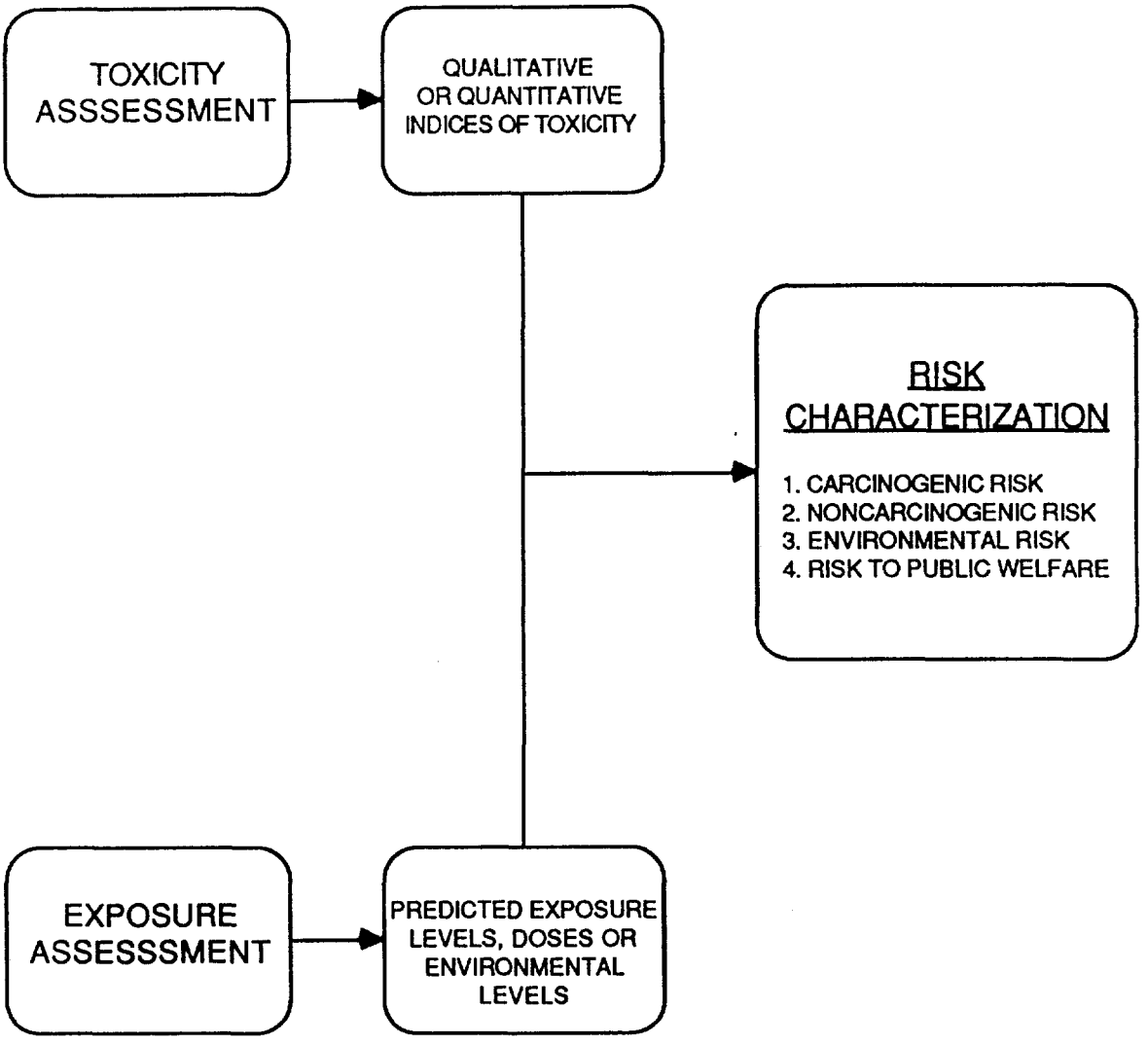


FIGURE 6. THE RISK CHARACTERIZATION PROCESS.



TABLES

TABLE 1.
Environmental Conservation and Chemical Corporation
Standard Parameters Used for Calculation of Dosage and Intake

Parameter	Adult	Child age 6-12	Child age 2-6
Physical Characteristics			
Average Body Weight	70 kg (1,2)	29 kg (3)	16 kg (3)
Average Surface Area	19400 cm2 (4)	10470 cm2 (4)	6980 cm2(4)
Activity Characteristics			
Amount of Air Breathed Daily	20 m3 (1)	11 m3 (1)	6 m3 (1)
Soil Ingested (Pica) Daily			1.0 g (1)
Frequency of Casual Contact to Surface Water	150 days/yr (1)	150 days/yr (1)	
Duration of Exposure to Surface Water	1 hr/day (1)	1 hr/day (1)	
Material Characteristics			
Dust Adherence (commercial potting soil)	1.45 mg/cm3 (1)		
Dust Adherence (mineral clay kaolin)	2.77 mg/cm3 (1)		
Transfer Ratio of Contaminant From Water to Air	1/10000 (3)		
Mass Flux Rate (water-based)	0.2-0.5 mg/cm2/hr (1)		

(1) Draft Superfund Exposure Assessment Manual
(2) *Draft Superfund Public Health Evaluation Manual*
(3) EPA/600/8-85/010
(4) PB84-213941

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Table 2.
PHYSICAL AND CHEMICAL PROPERTIES OF THE INDICATOR CHEMICALS

ENVIRONMENTAL CONSERVATION AND CHEMICAL CORPORATION

	Chloroform	1,1,2-Trichloro-ethane	1,1,1-Trichloro-ethane	Trichloro-ethene	Tetra-chloro-ethene	Ethyl-benzene	Toluene	Phenol	PCBs	Bis(2-ethylhexyl) phthalate	Di-n-butyl Phthalate	Diethyl Phthalate	Dimethyl phthalate
Molecular Weight, g/mol	119	133	133	131	166	106	92	94	328-376	391	278	222	144
Melting Point, ° C	-63.5	-36.5	-30	-87	-22.7	-95	-95	43		-50	-35	-40.5	0.0
Boiling Point, ° C	61.2	113.7	74.1	86.7	121.4	136.2	110.6	181.8	385-420	386.9	340	298.5	282
Density, g/ml	1.49	1.44	1.339	1.46	1.626	0.867	0.867	1.07	1.3-1.8	0.99	1.047	1.12	1.19
PARTITION COEFFICIENT													
Water Solubility, ppm (25 °C)	8.20E+03	4.50E+03	1.50E+03	1.10E+03	1.50E+02	1.52E+02	5.35E+02	9.30E+04	3.10E-02	4.00E-01	1.30E+01	8.96E+02	4.32E+03
Octanol-Water, log Kow	1.97	2.07	2.50	2.38	2.60	3.15	2.73	1.46	6.04	8.73	5.60	2.50	1.56
Sediment-Water, Koc	31	56	152	126	364	1100	300	14.2	5.30E+05	2.00E+09	1.70E+05	142	17.4
Microorganism-Water, Kb [(ug/g)/(mg/L)]	26	33	81	97	252	470	148	9.4	1.30E+06	2.30E+08	4.70E+04	107	16
VOLATILIZATION COEFFICIENTS													
Henry's Law Constant atm-m3/mol	2.87E-03	7.42E-04	1.44E-02	9.10E-03	2.59E-02	6.43E-03	6.37E-03	4.54E-07	7.40E-01	3.00E-07	2.82E-07	1.14E-06	2.15E-06
Vapor Pressure, mmHg (25 ° C)	151	30	123	57.9	17.8	7	28.1 (20°C)	0.341	4.1E-05	2E-07	0.00001	0.0035	0.00149(20°C)
Reaeration Rate Ratio KvC/Kvo	0.583	NAV	0.53	0.55	0.51	0.49	0.53	NAV	0.35	NAV	NAV	NAV	NAV

KEY:
1.00E-03 = 0.001
NAV - not applicable to volatilization calculations
NA - not applicable

References:
Verschueren, K., 1983
US DOT, 1986
US EPA, 1982

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Table 3.
Summary of Toxicological Information
For the Indicator Chemicals
Environmental Conservation and Chemical Corporation

Relevant Requirements, Criteria, Advisories and Guidelines		Chloroform	1,1,2-Trichloroethane	1,1,1-Trichloroethane	Trichloro-ethene	Tetrachloro-ethene	Ethylbenzene	Toluene	Phenol	PCBs	bis(2-Ethylhexyl) phthalate	Di-n-butyl phthalate	Diethyl phthalate	Dimethyl phthalate
EPA MCL		1.00E-01		2.00E-01	5.00E-03				3.50E+00	7.90E-06	7.00E-01	3.50E+00	3.50E+02	3.50E+02
(Proposed)							6.80E-01	2.00E+00	HBN	HBN	HBN	HBN	HBN	HBN
EPA Water Quality Criteria														
fish and drinking water		1.90E-04	6.00E-04	1.84E+01	2.70E-03	8.00E-04	1.40E+00	1.43E+01	3.50E+00	7.90E-08	1.50E+01	3.50E+01	3.50E+02	3.13E+02
fish only		1.57E-02	4.18E-02	1.03E+03	8.70E-02	8.85E-03	3.28E+00	4.24E+02	7.69E+02	7.90E-08	5.00E+01	1.54E+02	1.80E+03	2.90E+03
protection of aquatic life		1.24E+00	9.40E+00	2.19E+01	8.40E-01				2.56E+00	1.40E-05				
EPA Drinking Water Health Advisories														
1 day	10kg (70kg)	NA	NA	1.40E+02	NA	3.40E+01	2.10E+01	1.80E+01						
10 days	10kg (70kg)	NA	NA	3.50E+01	NA	3.40E+01	2.10E+00	6.00E+00						
chronic	10kg (70kg)	NA	NA	35.0(125)	NA	1.94(6.8)	3.40E+00	1.08E+01						
OSHA	8 hr TWA (mg/m3)	2.40E+02	4.50E+01	1.90E+03	5.40E+02	6.70E+02	4.35E+02	7.50E+02	1.90E+01		5.00E+00	5.00E+00		5.00E+00
ACGIH	8 hr TWA (mg/m3)	5.00E+01	4.50E+01	1.90E+03	2.70E+02	3.35E+02	4.35E+02	3.75E+02	1.90E+01	5.00E-01	5.00E+00	5.00E+00		5.00E+00
Noncarcinogenic Effects														
Risk Characterization														
Oral (mg/kg/day)														
	AIC	NA		5.40E-01	NA	2.00E-02	1.00E-01	3.00E-01	1.00E-01	NA	2.00E-02	1.00E-01	1.30E+01	1.00E-01
	AIS	NA		NA	NA	NA	9.70E-01	4.30E-01	1.00E-01	NA	NA			
	ADI	NA		3.00E-02	5.40E-01									
Inhalation (mg/kg/day)														
	AIC	NA		1.10E+01	NA	NA	NA	1.50E+00	2.00E-02	NA	NA			
	AIS	NA		6.30E+00	NA	NA	NA	1.50E+00	1.90E-01	NA	NA			
	ADI	NA		NA	NA									
Median Effective Dose (mg/day)														
Oral		NA		5.45E+03	9.50E+00	1.46E+03	7.24E+02	2.69E+03	5.98E+01	NA	NA	4.20E+02	2.99E+04	
Inhalation		NA		5.45E+03	1.05E+00	7.27E+03	7.24E+02	2.69E+03	8.02E+01	NA	NA	4.20E+02	2.99E+04	
Carcinogenic Effects														
Potency Factor [1/(mg/kg/day)]														
Oral		8.10E-02	5.73E-02	NA	1.10E-02	5.10E-02	NA	NA	NA	4.34E+00	6.84E-04	NA	NA	NA
Inhalation				NA	4.60E-03	1.70E-03	NA	NA	NA			NA	NA	NA
10% Effective Dose (mg/kg/day)														
Oral		5.08E-01	2.78E+00	NA	6.67E+00	3.23E+00	NA	NA	NA	5.00E-02	5.00E+01	NA	NA	NA
Inhalation		5.08E-01	2.78E+00	NA	6.67E+00	3.23E+00	NA	NA	NA	5.00E-02	5.00E+01	NA	NA	NA
Cancer Risk														
Inhalation at 1µg/m3(1)		1.00E-05	NA	3.00E-09	4.10E-06	1.70E-06	NA	NA	NA	1.20E-02	NA	NA	NA	NA
Water(E-6 Risk)GB		1.90E-01	6.00E-04	2.17E+01	2.70E-03	8.00E-04	NA	NA	NA	7.90E-08	NA	NA	NA	NA
Classification, EPA		B2	C	NC	B2	B2	NC	NC	NC	B2	B2	NC	NC	NC
Classification, IARC		2B	LE	LE	LE	3LE	NR	NR	NR	2B	NR	NR	NR	NR

Key:
HBN-Health Based Number
NA-Not Applicable
GB-EPA 1986 Water Quality Criteria
LE - Limited evidence of carcinogenicity
NR - Not ranked
(1) US EPA Office of Air and Radiation.

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Table 4.
Relative Importance of Processes Influencing Fate of the Indicator Chemicals at the Environmental Conservation and Chemical Corporation Site

CHEMICAL NAME	SORPTION	VOLATILIZATION	BIODEGRADATION	PHOTOLYSIS	HYDROLYSIS	BIOACCUMULATION	OXIDATION
Chloroform	-	+	?	-	+	-	+
1,1,2-Trichloroethane	-	+	-	-	+	-	+
1,1,1-Trichloroethane	-	+	-	-	+	-	+
Trichloroethene	-	+	?	-	+	-	?
Tetrachloroethene	-	+	?	-	+	-	+
Ethylbenzene	?	+	?	-	-	-	+
Toluene	+	+	?	-	-	-	+
Phenol	-	-	+	+	-	-	+
Polychlorinated Biphenyls (PCBs)	+	-	+	?	-	+	?
Bis(2-ethylhexyl)phthalate (DEHP)	+	-	+	-	-	+	-
Di-n-butyl Phthalate	+	-	+	-	-	+	-
Diethyl Phthalate	+	-	+	-	-	+	-
Dimethyl phthalate	+	-	+	-	-	+	-

KEY:
+ Could be important fate process
- Not likely to be an important fate process
? Importance of fate process uncertain or unknown

Reference:
Mills,W.B., et al, 1982.
Callahan, M.A., et al, 1979.

Table 5
Potential Exposure Pathways for the ECC Site

Transport Media	Source	Release Mechanism	Exposure Point	Exposure Route	Selected for Analysis
Air	On-site contaminated soil	Volatilization	On-site or off-site	Inhalation	Yes
	Contaminated surface water	Volatilization	On-site or off-site	Inhalation	Yes
	Contaminated groundwater	Volatilization during household use	Residential well	Inhalation	No - no such use expected
	Contaminated groundwater	Volatilization while showering/bathing	Residential well	Inhalation	No - no such use expected
	Contaminated soil	Fugitive dust generation	On-site or off-site	Inhalation	No - site capped or vegetated
Ground water	Contaminated soil	Leaching	Residential well	Ingestion Dermal Inhalation	No - no such use expected No - no such use expected No - no such use expected
Surface water	Contaminated soil	Run-off	Un-named ditch, Finley Creek	Ingestion Dermal Bioaccumulation	No - not a drinking water source Yes Yes
	Contaminated ground water	Surface water recharge	Finley Creek	Ingestion Dermal Bioaccumulation	No - not a drinking water source Yes Yes
Soil	Contaminated surface soils	Episodic overland flows	Nearest off-site residence	Dermal	No - site capped
	Contaminated surface soils			Dermal Ingestion	Yes Yes

Table 6.
Environmental Conservation and Chemical Corporation Site
Exposure Point Concentrations

Transport Media	Exposure Point	Indicator Chemical	Maximum Concentration (mg/L)	Average Concentration (mg/L)
Air	On-site (mg/m3)	Chloroform	1.22E-10	NA
		1,1,2-Trichloroethane	3.51E-08	NA
		1,1,1-Trichloroethane	1.42E-12	NA
		Trichloroethene	6.71E-10	NA
		Tetrachloroethene	5.70E-10	NA
		Ethylbenzene	1.32E-09	NA
		Toluene	3.41E-09	NA
		Phenol	1.16E-11	NA
		PCBs	4.74E-16	NA
		bis(2-Ethylhexyl)phthalate	0.00E+00	NA
		Di-n-butyl phthalate	0.00E+00	NA
		Diethyl phthalate	0.00E+00	NA
		Dimethyl phthalate	0.00E+00	NA
Soil	On-site (mg/l)	Chloroform	2.90E+00	9.55E-02
		1,1,2-Trichloroethane	5.50E-01	2.48E-02
		1,1,1-Trichloroethane	1.10E+03	1.18E+01
		Trichloroethene	1.10E+02	7.97E+00
		Tetrachloroethene	6.50E+02	2.62E+01
		Ethylbenzene	1.50E+03	6.91E+01
		Toluene	2.00E+03	1.21E+02
		Phenol	5.70E+02	1.92E+01
		PCBs	3.90E+01	1.40E+00
		bis(2-Ethylhexyl)phthalate	3.70E+02	1.82E+01
		Di-n-butyl phthalate	8.20E+00	1.48E+00
		Diethyl phthalate	9.00E+00	3.29E-01
		Dimethyl phthalate	1.30E+00	1.76E-01
Surface Water	Surface Water* (mg/l)	Chloroform	3.77E-10	5.16E-11
		1,1,2-Trichloroethane	0.00E+00	0.00E+00
		1,1,1-Trichloroethane	0.00E+00	0.00E+00
		Trichloroethene	0.00E+00	0.00E+00
		Tetrachloroethene	0.00E+00	0.00E+00
		Ethylbenzene	0.00E+00	0.00E+00
		Toluene	0.00E+00	0.00E+00
		Phenol	0.00E+00	0.00E+00
		PCBs	0.00E+00	0.00E+00
		bis(2-Ethylhexyl)phthalate	0.00E+00	0.00E+00
		Di-n-butyl phthalate	0.00E+00	0.00E+00
		Diethyl phthalate	0.00E+00	0.00E+00
		Dimethyl phthalate	0.00E+00	0.00E+00

* *Surface Water concentrations in the ditch were modeled and* where <1.00E-10, the effetive concentration was assumed zero.

NA - Not Applicable

Table 7.
Environmental Conservation and Chemical Corporation Site
Routes of Exposure Used in Calculation of Intakes

Exposure Scenario	Exposed Population	Routes of Exposure		
		Dermal	Ingestion	Inhalation
Occupational	Adult	Soil Contact Surface Water Contact	None	Daily Air
Residential	Child 2-6	Play in Soil	PICA	Household Air
	Child 6-12	Play in Soil Surface Water Contact	None	Household Air
	Adult	Soil Contact Surface Water Contact	None	Household Air

Table 8.
Environmental Conservation and Chemical Corporation Site
Characteristics of Subchronic/Chronic Exposure Scenarios

Route of Exposure	Media	Activity	Population	Subchronic Exposure Characteristics	Chronic Exposure Characteristics
Dermal	Soil	Casual Contact	Child age 2-6 Child age 6-12 Adult	Three exposure events (hands only) at average concentration or one event at highest conc., whichever is greatest; includes 100 mg of incidentally ingested soil	One exposure event (hands only) per day, 150 days per year, at average concentration; includes 100 mg of incidentally ingested soil
	Surface Water	Casual Contact	Child age 6-12 Adult	Three hours of exposure (20% of body) at average concentration or one hour at highest concentration, whichever is greatest	One hour of exposure (20% of body), 150 days per year, at average concentration
Ingestion	Soil	Pica	Child age 2-6	5 gram per day at average concentration or 1 gram at highest concentration, whichever is greatest	1 gram per day, 150 days per year, at average concentration
Inhalation	Combined Soil/ Surface Water Emission	Home	Child age 2-6 Child age 6-12	24 hours of exposure on-site at average predicted emission rate or 18 hr at highest predicted emission rate, whichever is greatest	18 hours of exposure, 365 days per year, on-site at average predicted emission rate
			Adult	24 hours of exposure on-site at average predicted emission rate or 16 hr at highest predicted emission rate, whichever is greatest	16 hours of exposure, 365 days per year, on-site at average predicted emission rate
		Occupational	Adult	8 hours of exposure on-site at highest predicted emission rate	8 hours of exposure, 250 days per year, on-site at average predicted emission rate

Table 9.
Environmental Conservation and Chemical Corporation Site
Calculation of Subchronic Daily Dermal Intakes

Exposure Scenario/ Exposed Population	Subpopulation	Indicator Chemical	Dermal Subchronic Daily Intakes		Total Dermal Subchronic Daily Intake (mg/kg)
			Soil Contact (mg/kg)	Surface Water (mg/kg)	
Occupational	Adult	Chloroform	7.83E-06	5.28E-15	7.83E-06
		1,1,2-Trichloroethane	1.49E-06	0.00E+00	1.49E-06
		1,1,1-Trichloroethane	2.97E-03	0.00E+00	2.97E-03
		Trichloroethene	2.97E-04	0.00E+00	2.97E-04
		Tetrachloroethene	1.76E-03	0.00E+00	1.76E-03
		Ethylbenzene	4.05E-03	0.00E+00	4.05E-03
		Toluene	5.40E-03	0.00E+00	5.40E-03
		Phenol	1.54E-03	0.00E+00	1.54E-03
		PCBs	1.05E-04	0.00E+00	1.05E-04
		bis(2-Ethylhexyl)phthalate	9.99E-04	0.00E+00	9.99E-04
		Di-n-butyl phthalate	2.21E-05	0.00E+00	2.21E-05
		Diethyl phthalate	2.43E-05	0.00E+00	2.43E-05
		Dimethyl phthalate	3.51E-06	0.00E+00	3.51E-06
Residential	Child 2-6	Chloroform	2.61E-05	NA	2.61E-05
		1,1,2-Trichloroethane	4.95E-06	NA	4.95E-06
		1,1,1-Trichloroethane	9.90E-03	NA	9.90E-03
		Trichloroethene	9.90E-04	NA	9.90E-04
		Tetrachloroethene	5.85E-03	NA	5.85E-03
		Ethylbenzene	1.35E-02	NA	1.35E-02
		Toluene	1.80E-02	NA	1.80E-02
		Phenol	5.13E-03	NA	5.13E-03
		PCBs	3.51E-04	NA	3.51E-04
		bis(2-Ethylhexyl)phthalate	3.33E-03	NA	3.33E-03
		Di-n-butyl phthalate	7.38E-05	NA	7.38E-05
		Diethyl phthalate	8.10E-05	NA	8.10E-05
		Dimethyl phthalate	1.17E-05	NA	1.17E-05
	Child 6-12	Chloroform	1.60E-05	6.79E-15	1.60E-05
		1,1,2-Trichloroethane	3.03E-06	0.00E+00	3.03E-06
		1,1,1-Trichloroethane	6.05E-03	0.00E+00	6.05E-03
		Trichloroethene	6.05E-04	0.00E+00	6.05E-04
		Tetrachloroethene	3.58E-03	0.00E+00	3.58E-03
		Ethylbenzene	8.25E-03	0.00E+00	8.25E-03
		Toluene	1.10E-02	0.00E+00	1.10E-02
		Phenol	3.14E-03	0.00E+00	3.14E-03
		PCBs	2.15E-04	0.00E+00	2.15E-04
		bis(2-Ethylhexyl)phthalate	2.04E-03	0.00E+00	2.04E-03
		Di-n-butyl phthalate	4.51E-05	0.00E+00	4.51E-05
		Diethyl phthalate	4.95E-05	0.00E+00	4.95E-05
		Dimethyl phthalate	7.15E-06	0.00E+00	7.15E-06
	Adult	Chloroform	7.83E-06	5.28E-15	7.83E-06
		1,1,2-Trichloroethane	1.49E-06	0.00E+00	1.49E-06
		1,1,1-Trichloroethane	2.97E-03	0.00E+00	2.97E-03
		Trichloroethene	2.97E-04	0.00E+00	2.97E-04
		Tetrachloroethene	1.76E-03	0.00E+00	1.76E-03
		Ethylbenzene	4.05E-03	0.00E+00	4.05E-03
		Toluene	5.40E-03	0.00E+00	5.40E-03
		Phenol	1.54E-03	0.00E+00	1.54E-03
		PCBs	1.05E-04	0.00E+00	1.05E-04
		bis(2-Ethylhexyl)phthalate	9.99E-04	0.00E+00	9.99E-04
		Di-n-butyl phthalate	2.21E-05	0.00E+00	2.21E-05
		Diethyl phthalate	2.43E-05	0.00E+00	2.43E-05
		Dimethyl phthalate	3.51E-06	0.00E+00	3.51E-06

Table 10.
Environmental Conservation and Chemical Corporation Site
Calculation of Subchronic Daily Ingestion Intakes

Exposure Scenario/ Exposed Population	Subpopulation	Indicator Chemical	Ingestion Subchronic Daily Intakes — Pica (mg/kg)	Total Ingestion Subchronic Daily Intake (mg/kg)
Occupational	Adult	Chloroform	NA	0.00E+00
		1,1,2-Trichloroethane	NA	0.00E+00
		1,1,1-Trichloroethane	NA	0.00E+00
		Trichloroethene	NA	0.00E+00
		Tetrachloroethene	NA	0.00E+00
		Ethylbenzene	NA	0.00E+00
		Toluene	NA	0.00E+00
		Phenol	NA	0.00E+00
		PCBs	NA	0.00E+00
		bis(2-Ethylhexyl)phthalate	NA	0.00E+00
		Di-n-butyl phthalate	NA	0.00E+00
		Diethyl phthalate	NA	0.00E+00
		Dimethyl phthalate	NA	0.00E+00
Residential	Child 2-6	Chloroform	1.81E-04	1.81E-04
		1,1,2-Trichloroethane	3.44E-05	3.44E-05
		1,1,1-Trichloroethane	6.88E-02	6.88E-02
		Trichloroethene	6.88E-03	6.88E-03
		Tetrachloroethene	4.06E-02	4.06E-02
		Ethylbenzene	9.38E-02	9.38E-02
		Toluene	1.25E-01	1.25E-01
		Phenol	3.56E-02	3.56E-02
		PCBs	2.44E-03	2.44E-03
		bis(2-Ethylhexyl)phthalate	2.31E-02	2.31E-02
		Di-n-butyl phthalate	5.13E-04	5.13E-04
		Diethyl phthalate	5.63E-04	5.63E-04
		Dimethyl phthalate	8.13E-05	8.13E-05
	Child 6-12	Chloroform	NA	0.00E+00
		1,1,2-Trichloroethane	NA	0.00E+00
		1,1,1-Trichloroethane	NA	0.00E+00
		Trichloroethene	NA	0.00E+00
		Tetrachloroethene	NA	0.00E+00
		Ethylbenzene	NA	0.00E+00
		Toluene	NA	0.00E+00
		Phenol	NA	0.00E+00
		PCBs	NA	0.00E+00
		bis(2-Ethylhexyl)phthalate	NA	0.00E+00
		Di-n-butyl phthalate	NA	0.00E+00
		Diethyl phthalate	NA	0.00E+00
		Dimethyl phthalate	NA	0.00E+00
	Adult	Chloroform	NA	0.00E+00
		1,1,2-Trichloroethane	NA	0.00E+00
		1,1,1-Trichloroethane	NA	0.00E+00
		Trichloroethene	NA	0.00E+00
		Tetrachloroethene	NA	0.00E+00
		Ethylbenzene	NA	0.00E+00
		Toluene	NA	0.00E+00
		Phenol	NA	0.00E+00
		PCBs	NA	0.00E+00
		bis(2-Ethylhexyl)phthalate	NA	0.00E+00
		Di-n-butyl phthalate	NA	0.00E+00
		Diethyl phthalate	NA	0.00E+00
		Dimethyl phthalate	NA	0.00E+00

Table 11.
Environmental Conservation and Chemical Corporation Site
Calculation of Subchronic Daily Inhalation Intakes

Exposure Scenario/ Exposed Population	Subpopulation	Indicator Chemical	Inhalation Subchronic Daily Intakes — Household Air (mg/kg)	Total Inhalation Subchronic Daily Intake (mg/kg)
Occupational	Adult	Chloroform	1.17E-11	1.17E-11
		1,1,2-Trichloroethane	3.37E-09	3.37E-09
		1,1,1-Trichloroethane	1.36E-13	1.36E-13
		Trichloroethene	6.44E-11	6.44E-11
		Tetrachloroethene	5.47E-11	5.47E-11
		Ethylbenzene	1.27E-10	1.27E-10
		Toluene	3.27E-10	3.27E-10
		Phenol	1.11E-12	1.11E-12
		PCBs	4.55E-17	4.55E-17
		bis(2-Ethylhexyl)phthalate	0.00E+00	0.00E+00
		Di-n-butyl phthalate	0.00E+00	0.00E+00
		Diethyl phthalate	0.00E+00	0.00E+00
		Dimethyl phthalate	0.00E+00	0.00E+00
Residential	Child 2-6	Chloroform	3.51E-11	3.51E-11
		1,1,2-Trichloroethane	1.01E-08	1.01E-08
		1,1,1-Trichloroethane	4.09E-13	4.09E-13
		Trichloroethene	1.93E-10	1.93E-10
		Tetrachloroethene	1.64E-10	1.64E-10
		Ethylbenzene	3.80E-10	3.80E-10
		Toluene	9.82E-10	9.82E-10
		Phenol	3.34E-12	3.34E-12
		PCBs	1.37E-16	1.37E-16
		bis(2-Ethylhexyl)phthalate	0.00E+00	0.00E+00
		Di-n-butyl phthalate	0.00E+00	0.00E+00
		Diethyl phthalate	0.00E+00	0.00E+00
		Dimethyl phthalate	0.00E+00	0.00E+00
	Child 6-12	Chloroform	3.29E-11	3.29E-11
		1,1,2-Trichloroethane	9.48E-09	9.48E-09
		1,1,1-Trichloroethane	3.83E-13	3.83E-13
		Trichloroethene	1.81E-10	1.81E-10
		Tetrachloroethene	1.54E-10	1.54E-10
		Ethylbenzene	3.56E-10	3.56E-10
		Toluene	9.21E-10	9.21E-10
		Phenol	3.13E-12	3.13E-12
		PCBs	1.28E-16	1.28E-16
		bis(2-Ethylhexyl)phthalate	0.00E+00	0.00E+00
		Di-n-butyl phthalate	0.00E+00	0.00E+00
		Diethyl phthalate	0.00E+00	0.00E+00
		Dimethyl phthalate	0.00E+00	0.00E+00
	Adult	Chloroform	2.34E-11	2.34E-11
		1,1,2-Trichloroethane	6.74E-09	6.74E-09
		1,1,1-Trichloroethane	2.73E-13	2.73E-13
		Trichloroethene	1.29E-10	1.29E-10
		Tetrachloroethene	1.09E-10	1.09E-10
		Ethylbenzene	2.53E-10	2.53E-10
		Toluene	6.55E-10	6.55E-10
		Phenol	2.23E-12	2.23E-12
		PCBs	9.10E-17	9.10E-17
		bis(2-Ethylhexyl)phthalate	0.00E+00	0.00E+00
		Di-n-butyl phthalate	0.00E+00	0.00E+00
		Diethyl phthalate	0.00E+00	0.00E+00
		Dimethyl phthalate	0.00E+00	0.00E+00

Table 12.
Environmental Conservation and Chemical Corporation Site
Calculation of Chronic Daily Dermal Intakes

Exposure Scenario/ Exposed Population	Subpopulation	Indicator Chemical	Dermal Chronic Daily Intakes		Total Dermal Chronic Daily Intake (mg/kg)
			Soil Contact (mg/kg)	Surface Water (mg/kg)	
Occupational	Adult	Chloroform	1.05E-07	2.84E-16	1.05E-07
		1,1,2-Trichloroethane	2.73E-08	0.00E+00	2.73E-08
		1,1,1-Trichloroethane	1.30E-05	0.00E+00	1.30E-05
		Trichloroethene	8.77E-06	0.00E+00	8.77E-06
		Tetrachloroethene	2.88E-05	0.00E+00	2.88E-05
		Ethylbenzene	7.60E-05	0.00E+00	7.60E-05
		Toluene	1.33E-04	0.00E+00	1.33E-04
		Phenol	2.11E-05	0.00E+00	2.11E-05
		PCBs	1.54E-06	0.00E+00	1.54E-06
		bis(2-Ethylhexyl)phthalate	2.00E-05	0.00E+00	2.00E-05
		Di-n-butyl phthalate	1.63E-06	0.00E+00	1.63E-06
		Diethyl phthalate	3.62E-07	0.00E+00	3.62E-07
		Dimethyl phthalate	1.94E-07	0.00E+00	1.94E-07
Residential	Child 2-6	Chloroform	3.53E-07	NA	3.53E-07
		1,1,2-Trichloroethane	9.18E-08	NA	9.18E-08
		1,1,1-Trichloroethane	4.37E-05	NA	4.37E-05
		Trichloroethene	2.95E-05	NA	2.95E-05
		Tetrachloroethene	9.69E-05	NA	9.69E-05
		Ethylbenzene	2.56E-04	NA	2.56E-04
		Toluene	4.48E-04	NA	4.48E-04
		Phenol	7.10E-05	NA	7.10E-05
		PCBs	5.18E-06	NA	5.18E-06
		bis(2-Ethylhexyl)phthalate	6.73E-05	NA	6.73E-05
		Di-n-butyl phthalate	5.48E-06	NA	5.48E-06
		Diethyl phthalate	1.22E-06	NA	1.22E-06
		Dimethyl phthalate	6.51E-07	NA	6.51E-07
	Child 6-12	Chloroform	2.20E-07	3.72E-16	2.20E-07
		1,1,2-Trichloroethane	5.70E-08	0.00E+00	5.70E-08
		1,1,1-Trichloroethane	2.71E-05	0.00E+00	2.71E-05
		Trichloroethene	1.83E-05	0.00E+00	1.83E-05
		Tetrachloroethene	6.03E-05	0.00E+00	6.03E-05
		Ethylbenzene	1.59E-04	0.00E+00	1.59E-04
		Toluene	2.78E-04	0.00E+00	2.78E-04
		Phenol	4.42E-05	0.00E+00	4.42E-05
		PCBs	3.22E-06	0.00E+00	3.22E-06
		bis(2-Ethylhexyl)phthalate	4.19E-05	0.00E+00	4.19E-05
		Di-n-butyl phthalate	3.40E-06	0.00E+00	3.40E-06
		Diethyl phthalate	7.57E-07	0.00E+00	7.57E-07
		Dimethyl phthalate	4.05E-07	0.00E+00	4.05E-07
	Adult	Chloroform	1.05E-07	2.84E-16	1.05E-07
		1,1,2-Trichloroethane	2.73E-08	0.00E+00	2.73E-08
		1,1,1-Trichloroethane	1.30E-05	0.00E+00	1.30E-05
		Trichloroethene	8.77E-06	0.00E+00	8.77E-06
		Tetrachloroethene	2.88E-05	0.00E+00	2.88E-05
		Ethylbenzene	7.60E-05	0.00E+00	7.60E-05
		Toluene	1.33E-04	0.00E+00	1.33E-04
		Phenol	2.11E-05	0.00E+00	2.11E-05
		PCBs	1.54E-06	0.00E+00	1.54E-06
		bis(2-Ethylhexyl)phthalate	2.00E-05	0.00E+00	2.00E-05
		Di-n-butyl phthalate	1.63E-06	0.00E+00	1.63E-06
		Diethyl phthalate	3.62E-07	0.00E+00	3.62E-07
		Dimethyl phthalate	1.94E-07	0.00E+00	1.94E-07

NA - Not Applicable

Table 13.
Environmental Conservation and Chemical Corporation Site
Calculation of Chronic Daily Ingestion Intakes

Exposure Scenario/ Exposed Population	Subpopulation	Indicator Chemical	Ingestion Chronic Daily Intakes — Pica (mg/kg)	Total Ingestion Chronic Daily Intake (mg/kg)
Occupational	Adult	Chloroform	NA	0.00E+00
		1,1,2-Trichloroethane	NA	0.00E+00
		1,1,1-Trichloroethane	NA	0.00E+00
		Trichloroethene	NA	0.00E+00
		Tetrachloroethene	NA	0.00E+00
		Ethylbenzene	NA	0.00E+00
		Toluene	NA	0.00E+00
		Phenol	NA	0.00E+00
		PCBs	NA	0.00E+00
		bis(2-Ethylhexyl)phthalate	NA	0.00E+00
		Di-n-butyl phthalate	NA	0.00E+00
		Diethyl phthalate	NA	0.00E+00
		Dimethyl phthalate	NA	0.00E+00
Residential	Child 2-6	Chloroform	2.45E-06	2.45E-06
		1,1,2-Trichloroethane	6.37E-07	6.37E-07
		1,1,1-Trichloroethane	3.03E-04	3.03E-04
		Trichloroethene	2.05E-04	2.05E-04
		Tetrachloroethene	6.73E-04	6.73E-04
		Ethylbenzene	1.78E-03	1.78E-03
		Toluene	3.11E-03	3.11E-03
		Phenol	4.93E-04	4.93E-04
		PCBs	3.60E-05	3.60E-05
		bis(2-Ethylhexyl)phthalate	4.68E-04	4.68E-04
		Di-n-butyl phthalate	3.80E-05	3.80E-05
		Diethyl phthalate	8.46E-06	8.46E-06
		Dimethyl phthalate	4.52E-06	4.52E-06
	Child 6-12	Chloroform	NA	0.00E+00
		1,1,2-Trichloroethane	NA	0.00E+00
		1,1,1-Trichloroethane	NA	0.00E+00
		Trichloroethene	NA	0.00E+00
		Tetrachloroethene	NA	0.00E+00
		Ethylbenzene	NA	0.00E+00
		Toluene	NA	0.00E+00
		Phenol	NA	0.00E+00
		PCBs	NA	0.00E+00
		bis(2-Ethylhexyl)phthalate	NA	0.00E+00
		Di-n-butyl phthalate	NA	0.00E+00
		Diethyl phthalate	NA	0.00E+00
		Dimethyl phthalate	NA	0.00E+00
	Adult	Chloroform	NA	0.00E+00
		1,1,2-Trichloroethane	NA	0.00E+00
		1,1,1-Trichloroethane	NA	0.00E+00
		Trichloroethene	NA	0.00E+00
		Tetrachloroethene	NA	0.00E+00
		Ethylbenzene	NA	0.00E+00
		Toluene	NA	0.00E+00
		Phenol	NA	0.00E+00
		PCBs	NA	0.00E+00
		bis(2-Ethylhexyl)phthalate	NA	0.00E+00
		Di-n-butyl phthalate	NA	0.00E+00
		Diethyl phthalate	NA	0.00E+00
		Dimethyl phthalate	NA	0.00E+00

NA - Not Applicable

Table 14.
Environmental Conservation and Chemical Corporation Site
Calculation of Chronic Daily Inhalation Intakes

Exposure Scenario/ Exposed Population	Subpopulation	Indicator Chemical	Inhalation Chronic Daily Intakes: Household Air (mg/kg)	Total Inhalation Chronic Daily Intake (mg/kg)
Occupational	Adult	Chloroform	0.00E+00	0.00E+00
		1,1,2-Trichloroethane	0.00E+00	0.00E+00
		1,1,1-Trichloroethane	0.00E+00	0.00E+00
		Trichloroethene	0.00E+00	0.00E+00
		Tetrachloroethene	0.00E+00	0.00E+00
		Ethylbenzene	0.00E+00	0.00E+00
		Toluene	0.00E+00	0.00E+00
		Phenol	0.00E+00	0.00E+00
		PCBs	0.00E+00	0.00E+00
		bis(2-Ethylhexyl)phthalate	0.00E+00	0.00E+00
		Di-n-butyl phthalate	0.00E+00	0.00E+00
		Diethyl phthalate	0.00E+00	0.00E+00
		Dimethyl phthalate	0.00E+00	0.00E+00
Residential	Child 2-6	Chloroform	0.00E+00	0.00E+00
		1,1,2-Trichloroethane	0.00E+00	0.00E+00
		1,1,1-Trichloroethane	0.00E+00	0.00E+00
		Trichloroethene	0.00E+00	0.00E+00
		Tetrachloroethene	0.00E+00	0.00E+00
		Ethylbenzene	0.00E+00	0.00E+00
		Toluene	0.00E+00	0.00E+00
		Phenol	0.00E+00	0.00E+00
		PCBs	0.00E+00	0.00E+00
		bis(2-Ethylhexyl)phthalate	0.00E+00	0.00E+00
		Di-n-butyl phthalate	0.00E+00	0.00E+00
		Diethyl phthalate	0.00E+00	0.00E+00
		Dimethyl phthalate	0.00E+00	0.00E+00
	Child 6-12	Chloroform	0.00E+00	0.00E+00
		1,1,2-Trichloroethane	0.00E+00	0.00E+00
		1,1,1-Trichloroethane	0.00E+00	0.00E+00
		Trichloroethene	0.00E+00	0.00E+00
		Tetrachloroethene	0.00E+00	0.00E+00
		Ethylbenzene	0.00E+00	0.00E+00
		Toluene	0.00E+00	0.00E+00
		Phenol	0.00E+00	0.00E+00
		PCBs	0.00E+00	0.00E+00
		bis(2-Ethylhexyl)phthalate	0.00E+00	0.00E+00
		Di-n-butyl phthalate	0.00E+00	0.00E+00
		Diethyl phthalate	0.00E+00	0.00E+00
		Dimethyl phthalate	0.00E+00	0.00E+00
	Adult	Chloroform	0.00E+00	0.00E+00
		1,1,2-Trichloroethane	0.00E+00	0.00E+00
		1,1,1-Trichloroethane	0.00E+00	0.00E+00
		Trichloroethene	0.00E+00	0.00E+00
		Tetrachloroethene	0.00E+00	0.00E+00
		Ethylbenzene	0.00E+00	0.00E+00
		Toluene	0.00E+00	0.00E+00
		Phenol	0.00E+00	0.00E+00
		PCBs	0.00E+00	0.00E+00
		bis(2-Ethylhexyl)phthalate	0.00E+00	0.00E+00
		Di-n-butyl phthalate	0.00E+00	0.00E+00
		Diethyl phthalate	0.00E+00	0.00E+00
		Dimethyl phthalate	0.00E+00	0.00E+00

Table 15.
ENVIRONMENTAL CONSERVATION AND CHEMICAL CORPORATION SITE

COMPARISON WITH POTENTIALLY APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS

PARAMETER	Surface Water Concentration Maximum (mg/L)	Surface Water Concentration Mean (mg/L)	Ambient Water Quality Criteria (C) Protection of Aquatic Life Freshwater Chronic (mg/L)
Chloroform	3.77E-10	5.16E-11	1.24E+00
1,1,1-Trichloroethane	0.00E+00	0.00E+00	
1,1,2-Trichloroethane	0.00E+00	0.00E+00	9.40E+00
Trichloroethene	0.00E+00	0.00E+00	2.19E+01
Tetrachloroethene	0.00E+00	0.00E+00	8.40E-01
Ethyl benzene	0.00E+00	0.00E+00	
Toluene	0.00E+00	0.00E+00	
Phenol	0.00E+00	0.00E+00	2.56E+00
PCB'S	0.00E+00	0.00E+00	1.40E-05
bis(2-Ethylhexyl) phthalate	0.00E+00	0.00E+00	
Di-n-butyl phthalate	0.00E+00	0.00E+00	
Diethyl phthalate	0.00E+00	0.00E+00	
Dimethyl phthalate	0.00E+00	0.00E+00	

All parameters below applicable guidance levels. The major contamination at the site is in the soil for which there is no federal or state guidance.

KEY

1.00E-03 = 0.001

REFERENCES

(C) Clean Water Act

Table 16.
Environmental Conservation and Chemical Corporation Site
Calculation of Subchronic Hazard Indices

Exposure Scenario/ Exposed Population	Population	Indicator Chemical	Route-Specific Subchronic Daily Intakes (mg/kg/day)			Subchronic Acceptable Intakes (mg/kg/day)		Hazard Indices
			Dermal	Ingestion	Inhalation	Oral	Inhalation	
Occupational	**Adult**	Chloroform	7.83E-06	NA	1.17E-11			
		1,1,2-Trichloroethane	1.49E-06	NA	3.37E-09			
		1,1,1-Trichloroethane	2.97E-03	NA	1.36E-13		1.10E+01	2.70E-04
		Trichloroethene	2.97E-04	NA	6.44E-11			
		Tetrachloroethene	1.76E-03	NA	5.47E-11			
		Ethylbenzene	4.05E-03	NA	1.27E-10	9.70E-01		4.18E-03
		Toluene	5.40E-03	NA	3.27E-10	4.30E-01	1.50E+00	1.26E-02
		Phenol	1.54E-03	NA	1.11E-12	1.00E-01	1.90E-01	1.54E-02
		PCBs	1.05E-04	NA	4.55E-17			
		bis(2-Ethylhexyl)phthalate	9.99E-04	NA	0.00E+00			
		Di-n-butyl phthalate	2.21E-05	NA	0.00E+00			
		Diethyl phthalate	2.43E-05	NA	0.00E+00			
		Dimethyl phthalate	3.51E-06	NA	0.00E+00			
						Total Subchronic Hazard =		3.24E-02
Residential	**Child 2-6**	Chloroform	2.61E-05	1.81E-04	3.51E-11			
		1,1,2-Trichloroethane	4.95E-06	3.44E-05	1.01E-08			
		1,1,1-Trichloroethane	9.90E-03	6.88E-02	4.09E-13		1.10E+01	6.33E-03
		Trichloroethene	9.90E-04	6.88E-03	1.93E-10			
		Tetrachloroethene	5.85E-03	4.06E-02	1.64E-10			
		Ethylbenzene	1.35E-02	9.38E-02	3.80E-10	9.70E-01		1.11E-01
		Toluene	1.80E-02	1.25E-01	9.82E-10	4.30E-01	1.50E+00	3.33E-01
		Phenol	5.13E-03	3.56E-02	3.34E-12	1.00E-01	1.90E-01	4.08E-01
		PCBs	3.51E-04	2.44E-03	1.37E-16			
		bis(2-Ethylhexyl)phthalate	3.33E-03	2.31E-02	0.00E+00			
		Di-n-butyl phthalate	7.38E-05	5.13E-04	0.00E+00			
		Diethyl phthalate	8.10E-05	5.63E-04	0.00E+00			
		Dimethyl phthalate	1.17E-05	8.13E-05	0.00E+00			
						Total Subchronic Hazard =		8.57E-01

Table 16. (Continued)
Environmental Conservation and Chemical Corporation Site
Calculation of Subchronic Hazard Indices

Exposure Scenario/ Exposed Population	Population	Indicator Chemical	Route-Specific Subchronic Daily Intakes (mg/kg/day)			Subchronic Acceptable Intakes (mg/kg/day)		Hazard Indices
			Dermal	Ingestion	Inhalation	Oral	Inhalation	
Residential	**Child 6-12**	Chloroform	1.60E-05	NA	3.29E-11			
		1,1,2-Trichloroethane	3.03E-06	NA	9.48E-09			
		1,1,1-Trichloroethane	6.05E-03	NA	3.83E-13		1.10E+01	5.50E-04
		Trichloroethene	6.05E-04	NA	1.81E-10			
		Tetrachloroethene	3.58E-03	NA	1.54E-10			
		Ethylbenzene	8.25E-03	NA	3.56E-10	9.70E-01		8.51E-03
		Toluene	1.10E-02	NA	9.21E-10	4.30E-01	1.50E+00	2.56E-02
		Phenol	3.14E-03	NA	3.13E-12	1.00E-01	1.90E-01	3.14E-02
		PCBs	2.15E-04	NA	1.28E-16			
		bis(2-Ethylhexyl)phthalate	2.04E-03	NA	0.00E+00			
		Di-n-butyl phthalate	4.51E-05	NA	0.00E+00			
		Diethyl phthalate	4.95E-05	NA	0.00E+00			
		Dimethyl phthalate	7.15E-06	NA	0.00E+00			
						Total Subchronic Hazard =		6.60E-02
	Adult	Chloroform	7.83E-06	NA	2.34E-11			
		1,1,2-Trichloroethane	1.49E-06	NA	6.74E-09			
		1,1,1-Trichloroethane	2.97E-03	NA	2.73E-13		1.10E+01	2.70E-04
		Trichloroethene	2.97E-04	NA	1.29E-10			
		Tetrachloroethene	1.76E-03	NA	1.09E-10			
		Ethylbenzene	4.05E-03	NA	2.53E-10	9.70E-01		4.18E-03
		Toluene	5.40E-03	NA	6.55E-10	4.30E-01	1.50E+00	1.26E-02
		Phenol	1.54E-03	NA	2.23E-12	1.00E-01	1.90E-01	1.54E-02
		PCBs	1.05E-04	NA	9.10E-17			
		bis(2-Ethylhexyl)phthalate	9.99E-04	NA	0.00E+00			
		Di-n-butyl phthalate	2.21E-05	NA	0.00E+00			
		Diethyl phthalate	2.43E-05	NA	0.00E+00			
		Dimethyl phthalate	3.51E-06	NA	0.00E+00			
						Total Subchronic Hazard =		3.24E-02

Table 17.
Environmental Conservation and Chemical Corporation Site
Calculation of Chronic Hazard Indices

Exposure Scenario/ Exposed Population	Population	Indicator Chemical	Route-Specific Chronic Daily Intakes (mg/kg/day)			Chronic Acceptable Intakes (mg/kg/day)		Hazard Indices
			Dermal	Ingestion	Inhalation	Oral	Inhalation	
Occupational	Adult	Chloroform	1.05E-07	NA	0.00E+00	1.00E-02		1.05E-05
		1,1,2-Trichloroethane	2.73E-08	NA	0.00E+00			
		1,1,1-Trichloroethane	1.30E-05	NA	0.00E+00	5.40E-01	6.30E+00	2.40E-05
		Trichloroethene	8.77E-06	NA	0.00E+00			
		Tetrachloroethene	2.88E-05	NA	0.00E+00	2.00E-02		1.44E-03
		Ethylbenzene	7.60E-05	NA	0.00E+00	1.00E-01		7.60E-04
		Toluene	1.33E-04	NA	0.00E+00	3.00E-01	1.50E+00	4.44E-04
		Phenol	2.11E-05	NA	0.00E+00	1.00E-01	2.00E-02	2.11E-04
		PCBs	1.54E-06	NA	0.00E+00			
		bis(2-Ethylhexyl)phthalate	2.00E-05	NA	0.00E+00	2.00E-02		1.00E-03
		Di-n-butyl phthalate	1.63E-06	NA	0.00E+00	1.00E-01		1.63E-05
		Diethyl phthalate	3.62E-07	NA	0.00E+00	1.30E+01		2.78E-08
		Dimethyl phthalate	1.94E-07	NA	0.00E+00			
						Total Chronic Hazard=		3.91E-03
Residential	Child 2-6	Chloroform	3.53E-07	2.45E-06	0.00E+00	1.00E-02		2.81E-04
		1,1,2-Trichloroethane	9.18E-08	6.37E-07	0.00E+00			
		1,1,1-Trichloroethane	4.37E-05	3.03E-04	0.00E+00	5.40E-01	6.30E+00	6.05E-04
		Trichloroethene	2.95E-05	2.05E-04	0.00E+00			
		Tetrachloroethene	9.69E-05	6.73E-04	0.00E+00	2.00E-02		3.85E-02
		Ethylbenzene	2.56E-04	1.78E-03	0.00E+00	1.00E-01		2.03E-02
		Toluene	4.48E-04	3.11E-03	0.00E+00	3.00E-01	1.50E+00	1.29E-02
		Phenol	7.10E-05	4.93E-04	0.00E+00	1.00E-01	2.00E-02	5.01E-03
		PCBs	5.18E-06	3.60E-05	0.00E+00			
		bis(2-Ethylhexyl)phthalate	6.73E-05	4.68E-04	0.00E+00	2.00E-02		2.68E-02
		Di-n-butyl phthalate	5.48E-06	3.80E-05	0.00E+00	1.00E-01		4.35E-04
		Diethyl phthalate	1.22E-06	8.46E-06	0.00E+00	1.30E+01		7.44E-07
		Dimethyl phthalate	6.51E-07	4.52E-06	0.00E+00			
						Total Chronic Hazard=		1.05E-01

Table 17. (Continued)
Environmental Conservation and Chemical Corporation Site
Calculation of Chronic Hazard Indices

Exposure Scenario/ Exposed Population	Population	Indicator Chemical	Route-Specific Chronic Daily Intakes (mg/kg/day)			Chronic Acceptable Intakes (mg/kg/day)		Hazard Indices
			Dermal	Ingestion	Inhalation	Oral	Inhalation	
Residential	Child 6-12	Chloroform	2.20E-07	NA	0.00E+00	1.00E-02		2.20E-05
		1,1,2-Trichloroethane	5.70E-08	NA	0.00E+00			
		1,1,1-Trichloroethane	2.71E-05	NA	0.00E+00	5.40E-01	6.30E+00	5.03E-05
		Trichloroethene	1.83E-05	NA	0.00E+00			
		Tetrachloroethene	6.03E-05	NA	0.00E+00	2.00E-02		3.01E-03
		Ethylbenzene	1.59E-04	NA	0.00E+00	1.00E-01		1.59E-03
		Toluene	2.78E-04	NA	0.00E+00	3.00E-01	1.50E+00	9.28E-04
		Phenol	4.42E-05	NA	0.00E+00	1.00E-01	2.00E-02	4.42E-04
		PCBs	3.22E-06	NA	0.00E+00			
		bis(2-Ethylhexyl)phthalate	4.19E-05	NA	0.00E+00	2.00E-02		2.09E-03
		Di-n-butyl phthalate	3.40E-06	NA	0.00E+00	1.00E-01		3.40E-05
		Diethyl phthalate	7.57E-07	NA	0.00E+00	1.30E+01		5.82E-08
		Dimethyl phthalate	4.05E-07	NA	0.00E+00			
						Total Chronic Hazard=		8.17E-03
	Adult	Chloroform	1.05E-07	NA	0.00E+00	1.00E-02		1.05E-05
		1,1,2-Trichloroethane	2.73E-08	NA	0.00E+00			
		1,1,1-Trichloroethane	1.30E-05	NA	0.00E+00	5.40E-01	6.30E+00	2.40E-05
		Trichloroethene	8.77E-06	NA	0.00E+00			
		Tetrachloroethene	2.88E-05	NA	0.00E+00	2.00E-02		1.44E-03
		Ethylbenzene	7.60E-05	NA	0.00E+00	1.00E-01		7.60E-04
		Toluene	1.33E-04	NA	0.00E+00	3.00E-01	1.50E+00	4.44E-04
		Phenol	2.11E-05	NA	0.00E+00	1.00E-01	2.00E-02	2.11E-04
		PCBs	1.54E-06	NA	0.00E+00			
		bis(2-Ethylhexyl)phthalate	2.00E-05	NA	0.00E+00	2.00E-02		1.00E-03
		Di-n-butyl phthalate	1.63E-06	NA	0.00E+00	1.00E-01		1.63E-05
		Diethyl phthalate	3.62E-07	NA	0.00E+00	1.30E+01		2.78E-08
		Dimethyl phthalate	1.94E-07	NA	0.00E+00			
						Total Chronic Hazard=		3.91E-03
						Total Lifetime Weighted Hazard=		1.02E-02

Table 18.
Environmental Conservation and Chemical Corporation Site
Calculation of Carcinogenic Risk

Exposure Scenario/ Exposed Population	Population	Indicator Chemical	Route-Specific Chronic Daily Intakes (mg/kg/day)			Carcinogenic Potency Factor (mg/kg/day)-1		Carcinogenic Risk
			Dermal	Ingestion	Inhalation	Oral	Inhalation	
Occupational	Adult	Chloroform	1.05E-07	NA	0.00E+00	8.10E-02		9E-09
		1,1,2-Trichloroethane	2.73E-08	NA	0.00E+00	5.73E-02		2E-09
		Trichloroethene	8.77E-06	NA	0.00E+00	1.10E-02	4.60E-03	1E-07
		Tetrachloroethene	2.88E-05	NA	0.00E+00	5.10E-02	1.70E-03	1E-06
		PCBs	1.54E-06	NA	0.00E+00	4.34E+00	6.11E+00	7E-06
		bis(2-Ethylhexyl)phthalate	2.00E-05	NA	0.00E+00	6.84E-04		1E-08
						Total Carcinogenic Risk =		8E-06
Residential	Child age 2-6	Chloroform	3.53E-07	2.45E-06	0.00E+00	8.10E-02		2E-07
		1,1,2-Trichloroethane	9.18E-08	6.37E-07	0.00E+00	5.73E-02		4E-08
		Trichloroethene	2.95E-05	2.05E-04	0.00E+00	1.10E-02	4.60E-03	3E-06
		Tetrachloroethene	9.69E-05	6.73E-04	0.00E+00	5.10E-02	1.70E-03	4E-05
		PCBs	5.18E-06	3.60E-05	0.00E+00	4.34E+00	6.11E+00	2E-04
		bis(2-Ethylhexyl)phthalate	6.73E-05	4.68E-04	0.00E+00	6.84E-04		4E-07
						Total Carcinogenic Risk =		2E-04
	Child age 6-12	Chloroform	2.20E-07	NA	0.00E+00	8.10E-02		2E-08
		1,1,2-Trichloroethane	5.70E-08	NA	0.00E+00	5.73E-02		3E-09
		Trichloroethene	1.83E-05	NA	0.00E+00	1.10E-02	4.60E-03	2E-07
		Tetrachloroethene	6.03E-05	NA	0.00E+00	5.10E-02	1.70E-03	3E-06
		PCBs	3.22E-06	NA	0.00E+00	4.34E+00	6.11E+00	1E-05
		bis(2-Ethylhexyl)phthalate	4.19E-05	NA	0.00E+00	6.84E-04		3E-08
						Total Carcinogenic Risk =		2E-05
	Adult	Chloroform	1.05E-07	NA	0.00E+00	8.10E-02		9E-09
		1,1,2-Trichloroethane	2.73E-08	NA	0.00E+00	5.73E-02		2E-09
		Trichloroethene	8.77E-06	NA	0.00E+00	1.10E-02	4.60E-03	1E-07
		Tetrachloroethene	2.88E-05	NA	0.00E+00	5.10E-02	1.70E-03	1E-06
		PCBs	1.54E-06	NA	0.00E+00	4.34E+00	6.11E+00	7E-06
		bis(2-Ethylhexyl)phthalate	2.00E-05	NA	0.00E+00	6.84E-04		1E-08
						Total Carcinogenic Risk =		8E-06
						Lifetime Weighted Carcinogenic Risk=		2E-05

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REFERENCES

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APPENDIX A

EPA MODIFICATION TO IARC APPROACH

APPENDIX A

Carcinogen risk assessment basically involves two steps; (1) the quantitative identification of potential carcinogens and (2) the quantitative assessment of level of risk which encompasses determination of carcinogenic potency and determination of exposure.

1. Identification of carcinogens: Evidence of possible carcinogenicity in humans comes primarily from two sources: long-term animal tests and epidemiological investigations. Results from these studies are supplemented with information from short-term tests, pharmacokinetic studies, comparative metabolism studies, structural-activity relationships, and other relevant information sources. When judging qualitative evidence of carcinogenicity, EPA as well as the IARC have adopted a policy of "weight-of-evidence" meaning that the quality and adequacy of all relevant data on responses induced by a possible carcinogen using different procedures will be considered. There are three major steps in determining the weight-of-evidence for carcinogenicity:

   1. characterization of the evidence from human studies and from animal studies individually,

   2. combination of the two types of data into a final indication of overall weight-of-evidence for human carcinogenicity, and

   3. evaluation of all supportive information to determine if the overall weight-of-evidence should be modified.

The EPA classification system for chemical carcinogens modeled after the one developed by the International Agency for Research on Cancer (IARC) includes: Group A - carcinogenic to humans; Group B - "probably"carcinogenic to humans; this category includes agents for which the evidence of human carcinogenicity from epidemiologic studies ranges from almost "sufficient" to "inadequate". To reflect this range, the category is divided into higher (Group B1) and lower (Group B2) degrees of evidence. Usually, category B1 is reserved for agents for which there is at least limited evidence of carcinogenicity to humans from epidemiologic studies. Group C - "possibly" carcinogenic to humans; Group D - cannot be classified as to human carcinogenicity due to inadequate animal evidence of carcinogenicity; and Group E - no evidence of carcinogenicity for humans.

The IARC system is virtually identical to the EPA scheme with a Group 1 - substances carcinogenic to humans; Group 2 - probable human carcinogens featuring the same subdivisions 2A and 2B. However, there is no group corresponding to EPA's group C. Instead, IARC uses a Group 3 - compounds that cannot be classified as to its carcinogenicity to humans (corresponding to EPA group D).

In spite of being based on a common philosophy and using virtually the same data, the IARC as well as the competent authorities within the European Communities have come to different conclusions than the US EPA as to the assessment of the potential carcinogenicity of several compounds, e.g., some halogenated hydrocarbons. This difference of opinion mostly derives from different interpretation of animal data, in particular, the relevance of liver tumors in rodents.

2. Quantification of carcinogenic risk: The second phase in carcinogen assessment involves the quantification of risk. Since experimental studies of carcinogenic effects are not feasible at the low exposure levels usually encountered, various mathematical models have to be used for extrapolation from the high doses used in animal bioassays down to the dosages of interest in connection with exposure to ambient environmental concentrations. Since the resolution power of animal experiments like the NTP bioassays is not adequate for precise elaboration of the dose-response curve, this extrapolation is associated with a level of uncertainty which may amount to orders of magnitude. Given the well-known differences in carcinogenic response between species - or even between strains of the same species - it is obvious that additional uncertainties will be introduced when making quantitative extrapolations, i.e., rodent to man.

Among various proposed models for quantitative extrapolation EPA has recommended the use of a linearized multistage model "unless there is evidence on carcinogenesis mechanisms or other biological evidence that indicates the greater suitability of an alternative extrapolation model, or there is statistical or biological evidence that excludes the use of the linearized multistage model." (FR, Vol. 49, Nov. 23, 1984, p.46298). The carcinogenic potency of a chemical is often expressed in terms of a potency factor which is the upper 95 percent confidence limit on the probability of response per unit intake (mg/kg etc.) of a chemical over a lifetime. EPA's Carcinogen Assessment Group (CAG) has evaluated more than fifty-four chemicals as suspect human carcinogens and developed relative carcinogenic potency factors for each chemical.

The EPA has made the following modifications of the IARC approach to classifying human and animal studies. For human studies:

1. The observation of a statistically significant association between an agent and life threatening benign tumors in humans is included in the evaluations of risk to humans.

2. A "no evidence" category is added. This category indicates that no association was found between exposure and increased risk of cancer in well-conducted, well-designed, independent analytical epidemologic studies.

For animal studies:

1. An increased incident of combined benign and malignant tumors will be considered to provide sufficient evidence of carcinogenicity if the other criteria defining the "sufficient" category of evidence are met.

2. An increased incident of benign tumors alone as "limited" evidence of carcinogenicity is added.

3. Under specific circumstances, such as the production of neoplasms that occur with high spontaneous background incident, the evidence may be decreased to "limited" if warranted.

4. A "no evidence" category is also added.

Agents that are judged to be in the EPA Weight-of-Evidence stratification Groups A and B are to be regarded as suitable for quantitative risk assessments. The appropriateness of quantifying the risks from agents in Group C, specifically agents that are at the boundary of Group C and D, would be judged on a case-by-case basis. Agents that are judged to be in Groups D and E should generally not be evaluated using quantitative risk assessments.

Evidence of carcinogenicity from human studies comes from three main sources:

1. case reports of individual cancer patients who were exposed to the agent(s)

2. descriptive epidemological studies

3. analytical epidemologic (case control and cohort) studies

Five criteria must be met before a causal association can be inferred between exposure and cancer in humans:

1. There is no identified bias which can explain the association,

2. The possibility of confounding has been considered and ruled out as explaining the association,

3. The association is unlikely to be due to chance. The degrees of evidence for carcinogenicity from studies in humans can be categorized by:

    a. sufficient evidence of carcinogenicity, which indicates that there is a causal relationship between the agent and human cancer

    b. limited evidence of carcinogenicity, which indicates that a causal interpretation is credible

    c. inadequate evidence

        i. there were few pertinent data, or

        ii. the available studies, while showing evidence of association, did not exclude chance, bias or confounding

4. No evidence, and

5. No data.

Assessment of evidence for carcinogenicity from studies in experimental animals are classified into five groups.

1. Sufficient evidence of carcinogenicity, which indicates an incident of malignant tumors or combined malignant and benign tumors:

    a. in multiple species or strains; or

    b. in multiple experiments (preferably with different routes of administration or using different dose levels); or

    c. to an unusual degree with regard to incidence, site or type of tumor, or age at onset.

2. Limited evidence of carcinogenicity.

a. studies involve a single species, strain, or experiment; or

b. the experiments are restricted by inadequate dose levels, inadequate duration of exposure to the agent, inadequate period of follow-up, poor survival, too few animals, or inadequate reporting; or

c. an increase in the incident of benign tumors only.

3. Inadequate evidence.

4. No evidence.

5. No data.

The categorization of overall evidence of carcinogenicity is subdivided into five groups.

Group A: Human carcinogens are used only when there is sufficient evidence from epidemologic studies to support the causal association between exposure to agent(s) and cancer.

Group B: Probable human carcinogens include agents for which the evidence of human carcinogenicity from epidemologic studies ranges from almost "sufficient" to "inadequate". B1 is reserved for agents for which there is at least limited evidence of carcinogenicity to humans from epidemologic studies. The agents for which there is inadequate evidence from human studies but sufficient evidence from animal studies would usually result in a classification of B2.

Group C: Possible human carcinogens are used for agents with limited evidence of carcinogenicity in animals in the absence of human data. It includes a wide variety of evidence:

a. definitive malignant tumor response in a single well-conducted study,

b. marginal tumor responses in studies having inadequate design for reporting,

c. benign but not malignant tumors with an agent showing no response in a variety of short-term tests for mutagenicity, and

d. marginal responses in a tissue known to have a high and variable background rate.

Group D: Not classified is used for agent(s) with inadequate animal evidence of carcinogenicity.

Group E: No evidence of carcinogenicity for humans is used for agent(s) that shows no evidence for carcinogenicity in at least two adequate animal studies in different species or in both epidemologic animal studies.

The text for the general weight-of-evidence discussion is taken from proposed guidelines for carcinogen risk assessment (USEPA, 1984c).

The Carcinogen Assessment Group (CAG) has evaluated fifty-four chemicals as suspect human carcinogens and developed relative carcinogenic potency factors for each chemical. The ranking of potency indices is subjected to the uncertainty of comparing different routes of exposure and a number of different species. These indices are based on estimates of low dose risk using linear multistage extrapolation from the observed range. As stated in the Superfund Public Health Evaluation Manual, this is only valid at low risk levels. For sites where chemical intakes may be large, application of the linear multistage model assuming linearity may not be valid.

APPENDIX B

SUPPORTING INFORMATION TO FATE AND TRANSPORT CALCULATIONS

TABLE B-1
GROUND WATER
TRAVEL PARAMETERS

INDICATOR CHEMICAL	Koc	Kp	R	VELOCITY ft/yr	YEARS TO DITCH
CHLOROFORM	38.73	0.05	2.05	1.997	50
1,1,1-TRICHLOROETHANE	57.59	0.08	2.57	1.598	63
1,1,2-TRICHLOROETHANE	56.87	0.08	2.55	1.610	62
TRICHLOROETHENE	123.56	0.17	4.36	0.940	106
TETRACHLOROETHENE	82.00	0.11	3.23	1.269	79
TOLUENE	396.78	0.54	11.79	0.348	287
ETHYLBENZENE	827.22	1.13	23.50	0.174	575
PHENOL	25.97	0.04	1.71	2.403	42
BIS(2-ETHYLHEXYL)PHTHALATE	3,778.60	5.14	103.78	0.040	2,500
DI-N-BUTYLPHTHALATE	1,064.95	1.45	29.97	0.137	730
DIETHYLPHTHALATE	230.56	0.31	7.27	0.564	177
DIMETHYLPHTHALTE	40.32	0.05	2.10	1.955	51
PCB AROCLOR 1260	17,510.58	23.81	477.29	0.009	11,111

ECC RISK ASSESSMENT
SEPT. 30. 1987

CHLOROFORM			YEAR	INITIAL CONC. ppb	C(LT) ppb	C(GWT) ppb	YEAR	C(RTI) ppb
CONCENTRATION	ppm	0.0955	0.0	9.6E+01	1.8E+01	4.5E+00	50	1.3E-07
Di		0.08875	1.0		1.4E+01	3.4E+00	51	1.0E-07
p		160	2.0		1.0E+01	2.6E+00	52	7.5E-08
MWi		119.38	3.0		7.8E+00	2.0E+00	53	5.7E-08
Csi	pMWi/62.3*293	1.0E-03	4.0		5.9E+00	1.5E+00	54	4.3E-08
A		10,000	5.0		4.5E+00	1.1E+00	55	3.3E-08
Pt		0.35	6.0		3.4E+00	8.5E-01	56	2.5E-08
Pt^(4/3)		2.5E-01	7.0		2.6E+00	6.4E-01	57	1.9E-08
dsc	30.48*2.5	7.6E+01	8.0		1.9E+00	4.8E-01	58	1.4E-08
k(1v)		4.7E-02	9.0		1.5E+00	3.7E-01	59	1.1E-08
HALF-LIFE	YEARS	2	10.0		1.1E+00	2.8E-01	60	8.0E-09
LN(2)		6.9E-01	11.0		8.4E-01	2.1E-01	61	6.1E-09
k(1h)		3.5E-01	12.0		6.3E-01	1.6E-01	62	4.6E-09
kT(1v+1h)		3.9E-01	13.0		4.8E-01	1.2E-01	63	3.5E-09
SOLUBILTY	mg/l	8,000	14.0		3.6E-01	9.0E-02	64	2.6E-09
LENGTH	FEET	100	15.0		2.7E-01	6.8E-02	65	2.0E-09
VELOCITY	FT/YR	1.997	16.0		2.1E-01	5.2E-02	66	1.5E-09
PART. COEFF		0.06	17.0		1.6E-01	3.9E-02	67	1.1E-09
(t*)i	YEARS	50	18.0		1.2E-01	3.0E-02	68	8.6E-10
C(RTI)/c(gwti)		2.9E-08	19.0		8.9E-02	2.2E-02	69	6.5E-10
			20.0		6.8E-02	1.7E-02	70	4.9E-10
			22.5		3.4E-02	8.4E-03	73	2.4E-10
			25.0		1.7E-02	4.2E-03	75	1.2E-10
			27.5		8.3E-03	2.1E-03	78	6.0E-11
			30.0		4.1E-03	1.0E-03	80	3.0E-11
			32.5		2.0E-03	5.1E-04	83	1.5E-11
			35.0		1.0E-03	2.5E-04	85	7.4E-12
			37.5		5.1E-04	1.3E-04	88	3.7E-12
			40.0		2.5E-04	6.3E-05	90	1.8E-12
			42.5		1.2E-04	3.1E-05	93	9.1E-13
			45.0		6.2E-05	1.6E-05	95	4.5E-13
			47.5		3.1E-05	7.7E-06	98	2.2E-13
			50.0		1.5E-05	3.8E-06	100	1.1E-13
			52.5		7.6E-06	1.9E-06	103	5.5E-14
			55.0		3.8E-06	9.5E-07	105	2.7E-14
			57.5		1.9E-06	4.7E-07	108	1.4E-14
			60.0		9.3E-07	2.3E-07	110	6.8E-15
			62.5		4.6E-07	1.2E-07	113	3.4E-15
			65.0		2.3E-07	5.8E-08	115	1.7E-15
			67.5		1.1E-07	2.9E-08	118	8.3E-16
			70.0		5.7E-08	1.4E-08	120	4.1E-16

ECC RISK ASSESSMENT
SEPT. 30, 1987

1,1,1-TRICHLOROETHANE			YEAR	INITIAL CONC. ppb	C(LT) ppb	C(GWT) ppb	YEAR	C(RTI) ppb
CONCENTRATION	ppm	11.8	0.0	1.2E+04	4.6E+02	1.2E+02	63	4.4E-08
Di		0.07971	1.0		3.5E+02	8.8E+01	64	3.3E-08
p		100	2.0		2.7E+02	6.8E+01	65	2.6E-08
MWi		133.41	3.0		2.1E+02	5.2E+01	66	2.0E-08
Csi	pMWi/62.3*293	7.3E-04	4.0		1.6E+02	4.0E+01	67	1.5E-08
A		10,000	5.0		1.2E+02	3.0E+01	68	1.1E-08
Pt		0.35	6.0		9.3E+01	2.3E+01	69	8.8E-09
Pt^(4/3)		2.5E-01	7.0		7.1E+01	1.8E+01	70	6.7E-09
dsc	30.48*2.5	7.6E+01	8.0		5.4E+01	1.4E+01	71	5.2E-09
k(1v)		3.0E-02	9.0		4.2E+01	1.0E+01	72	3.9E-09
HALF-LIFE	YEARS	2	10.0		3.2E+01	8.0E+00	73	3.0E-09
LN(2)		6.9E-01	11.0		2.4E+01	6.1E+00	74	2.3E-09
k(1h)		3.5E-01	12.0		1.9E+01	4.7E+00	75	1.8E-09
kT(1v+1h)		3.8E-01	13.0		1.4E+01	3.6E+00	76	1.4E-09
SOLUBILITY	mg/l	4,400	14.0		1.1E+01	2.7E+00	77	1.0E-09
LENGTH	FEET	100	15.0		8.4E+00	2.1E+00	78	7.9E-10
VELOCITY	FT/YR	1.598	16.0		6.4E+00	1.6E+00	79	6.1E-10
PART. COEFF		0.35	17.0		4.9E+00	1.2E+00	80	4.7E-10
(t*)i	YEARS	63	18.0		3.8E+00	9.4E-01	81	3.6E-10
C(RTI)/c(gwti)		3.8E-10	19.0		2.9E+00	7.2E-01	82	2.7E-10
			20.0		2.2E+00	5.5E-01	83	2.1E-10
			22.5		1.1E+00	2.8E-01	85	1.1E-10
			25.0		5.8E-01	1.4E-01	88	5.5E-11
			27.5		3.0E-01	7.4E-02	90	2.8E-11
			30.0		1.5E-01	3.8E-02	93	1.4E-11
			32.5		7.8E-02	2.0E-02	95	7.4E-12
			35.0		4.0E-02	1.0E-02	98	3.8E-12
			37.5		2.1E-02	5.1E-03	100	1.9E-12
			40.0		1.1E-02	2.6E-03	103	1.0E-12
			42.5		5.4E-03	1.4E-03	105	5.1E-13
			45.0		2.8E-03	6.9E-04	108	2.6E-13
			47.5		1.4E-03	3.6E-04	110	1.3E-13
			50.0		7.3E-04	1.8E-04	113	6.9E-14
			52.5		3.7E-04	9.3E-05	115	3.5E-14
			55.0		1.9E-04	4.8E-05	118	1.8E-14
			57.5		9.8E-05	2.5E-05	120	9.3E-15
			60.0		5.0E-05	1.3E-05	123	4.8E-15
			62.5		2.6E-05	6.5E-06	125	2.4E-15
			65.0		1.3E-05	3.3E-06	128	1.3E-15
			67.5		6.8E-06	1.7E-06	130	6.4E-16
			70.0		3.5E-06	8.7E-07	133	3.3E-16

ECC RISK ASSESSMENT
SEPT. 30, 1987

1,1,2-TRICHLOROETHANE			YEAR	INITIAL CONC. ppb	C(LT) ppb	C(GWT) ppb	YEAR	C(RTI) ppb
CONCENTRATION	ppm	0.0248	0.0	2.5E+01	5.8E+00	1.5E+00	62	6.5E-10
Di		0.07971	1.0		4.5E+00	1.1E+00	63	5.1E-10
p		19	2.0		3.5E+00	8.8E-01	64	4.0E-10
MWi		133.41	3.0		2.8E+00	6.9E-01	65	3.1E-10
Csi	pMWi/62.3*293	1.4E-04	4.0		2.1E+00	5.4E-01	66	2.4E-10
A		10,000	5.0		1.7E+00	4.2E-01	67	1.9E-10
Pt		0.35	6.0		1.3E+00	3.3E-01	68	1.5E-10
Pt^(4/3)		2.5E-01	7.0		1.0E+00	2.5E-01	69	1.1E-10
dsc	30.48*2.5	7.6E+01	8.0		7.9E-01	2.0E-01	70	8.8E-11
k(1v)		5.6E-03	9.0		6.1E-01	1.5E-01	71	6.9E-11
HALF-LIFE	YEARS	2	10.0		4.8E-01	1.2E-01	72	5.4E-11
LN(2)		6.9E-01	11.0		3.7E-01	9.3E-02	73	4.2E-11
k(1h)		3.5E-01	12.0		2.9E-01	7.3E-02	74	3.2E-11
kT(1v+1h)		3.5E-01	13.0		2.3E-01	5.7E-02	75	2.5E-11
SOLUBILITY	mg/l	4,500	14.0		1.8E-01	4.4E-02	76	2.0E-11
LENGTH	FEET	100	15.0		1.4E-01	3.4E-02	77	1.5E-11
VELOCITY	FT/YR	1.61	16.0		1.1E-01	2.7E-02	78	1.2E-11
PART. COEFF		0.015	17.0		8.3E-02	2.1E-02	79	9.3E-12
(t*)i	YEARS	62	18.0		6.5E-02	1.6E-02	80	7.2E-12
C(RTI)/c(gwti)		4.5E-10	19.0		5.0E-02	1.3E-02	81	5.6E-12
			20.0		3.9E-02	9.8E-03	82	4.4E-12
			22.5		2.1E-02	5.3E-03	85	2.4E-12
			25.0		1.1E-02	2.8E-03	87	1.3E-12
			27.5		6.0E-03	1.5E-03	90	6.7E-13
			30.0		3.2E-03	8.0E-04	92	3.6E-13
			32.5		1.7E-03	4.3E-04	95	1.9E-13
			35.0		9.2E-04	2.3E-04	97	1.0E-13
			37.5		4.9E-04	1.2E-04	100	5.5E-14
			40.0		2.6E-04	6.6E-05	102	3.0E-14
			42.5		1.4E-04	3.5E-05	105	1.6E-14
			45.0		7.6E-05	1.9E-05	107	8.5E-15
			47.5		4.0E-05	1.0E-05	110	4.5E-15
			50.0		2.2E-05	5.4E-06	112	2.4E-15
			52.5		1.2E-05	2.9E-06	115	1.3E-15
			55.0		6.2E-06	1.6E-06	117	6.9E-16
			57.5		3.3E-06	8.3E-07	120	3.7E-16
			60.0		1.8E-06	4.4E-07	122	2.0E-16
			62.5		9.5E-07	2.4E-07	125	1.1E-16
			65.0		5.1E-07	1.3E-07	127	5.7E-17
			67.5		2.7E-07	6.8E-08	130	3.0E-17
			70.0		1.5E-07	3.6E-08	132	1.6E-17

ECC RISK ASSESSMENT
SEPT. 30, 1987

TRICHLOROETHENE			YEAR	INITIAL CONC. ppb	C(LT) ppb	C(GWT) ppb	YEAR	C(RTI) ppb
CONCENTRATION	ppm	7.97	0.0	8.0E+03	2.3E+02	5.7E+01	106	5.5E-15
Di		0.08122	1.0		1.8E+02	4.4E+01	107	4.3E-15
p		20	2.0		1.4E+02	3.4E+01	108	3.3E-15
MWi		131.50	3.0		1.1E+02	2.7E+01	109	2.6E-15
Csi	pMWi/62.3*293	1.4E-04	4.0		8.3E+01	2.1E+01	110	2.0E-15
A		10,000	5.0		6.5E+01	1.6E+01	111	1.6E-15
Pt		0.35	6.0		5.1E+01	1.3E+01	112	1.2E-15
Pt^(4/3)		2.5E-01	7.0		3.9E+01	9.8E+00	113	9.5E-16
dsc	30.48*2.5	7.6E+01	8.0		3.1E+01	7.7E+00	114	7.4E-16
k(1v)		6.0E-03	9.0		2.4E+01	6.0E+00	115	5.8E-16
HALF-LIFE	YEARS	2	10.0		1.9E+01	4.6E+00	116	4.5E-16
LN(2)		6.9E-01	11.0		1.4E+01	3.6E+00	117	3.5E-16
k(1h)		3.5E-01	12.0		1.1E+01	2.8E+00	118	2.7E-16
kT(1v+1h)		3.5E-01	13.0		8.8E+00	2.2E+00	119	2.1E-16
SOLUBILITY	mg/l	1,100	14.0		6.8E+00	1.7E+00	120	1.7E-16
LENGTH	FEET	100	15.0		5.3E+00	1.3E+00	121	1.3E-16
VELOCITY	FT/YR	0.94	16.0		4.1E+00	1.0E+00	122	1.0E-16
PART. COEFF		0.175	17.0		3.2E+00	8.1E-01	123	7.8E-17
(t*)i	YEARS	106	18.0		2.5E+00	6.3E-01	124	6.1E-17
C(RTI)/c(gwti)		9.7E-17	19.0		2.0E+00	4.9E-01	125	4.7E-17
			20.0		1.5E+00	3.8E-01	126	3.7E-17
			22.5		8.1E-01	2.0E-01	129	2.0E-17
			25.0		4.4E-01	1.1E-01	131	1.1E-17
			27.5		2.3E-01	5.8E-02	134	5.6E-18
			30.0		1.2E-01	3.1E-02	136	3.0E-18
			32.5		6.7E-02	1.7E-02	139	1.6E-18
			35.0		3.6E-02	8.9E-03	141	8.6E-19
			37.5		1.9E-02	4.8E-03	144	4.6E-19
			40.0		1.0E-02	2.5E-03	146	2.5E-19
			42.5		5.4E-03	1.4E-03	149	1.3E-19
			45.0		2.9E-03	7.3E-04	151	7.0E-20
			47.5		1.6E-03	3.9E-04	154	3.8E-20
			50.0		8.3E-04	2.1E-04	156	2.0E-20
			52.5		4.5E-04	1.1E-04	159	1.1E-20
			55.0		2.4E-04	6.0E-05	161	5.8E-21
			57.5		1.3E-04	3.2E-05	164	3.1E-21
			60.0		6.8E-05	1.7E-05	166	1.6E-21
			62.5		3.6E-05	9.1E-06	169	8.8E-22
			65.0		2.0E-05	4.9E-06	171	4.7E-22
			67.5		1.0E-05	2.6E-06	174	2.5E-22
			70.0		5.6E-06	1.4E-06	176	1.3E-22

ECC RISK ASSESSMENT
SEPT. 30, 1987

TETRACHLOROETHENE			YEAR	INITIAL CONC. ppb	C(LT) ppb	C(GWT) ppb	YEAR	C(RTI) ppb
CONCENTRATION	ppm	26.2	0.0	2.6E+04	7.1E+02	1.8E+02	79	2.4E-10
Di		0.07294	1.0		5.6E+02	1.4E+02	80	1.9E-10
p		5	2.0		4.4E+02	1.1E+02	81	1.5E-10
MWi		165.83	3.0		3.4E+02	8.5E+01	82	1.2E-10
Csi	pMWi/62.3*293	4.5E-05	4.0		2.7E+02	6.6E+01	83	9.1E-11
A		10,000	5.0		2.1E+02	5.2E+01	84	7.1E-11
Pt		0.35	6.0		1.6E+02	4.0E+01	85	5.5E-11
Pt^(4/3)		2.5E-01	7.0		1.3E+02	3.2E+01	86	4.3E-11
dsc	30.48*2.5	7.6E+01	8.0		9.9E+01	2.5E+01	87	3.4E-11
k(1v)		1.7E-03	9.0		7.7E+01	1.9E+01	88	2.6E-11
HALF-LIFE	YEARS	2	10.0		6.0E+01	1.5E+01	89	2.1E-11
LN(2)		6.9E-01	11.0		4.7E+01	1.2E+01	90	1.6E-11
k(1h)		3.5E-01	12.0		3.7E+01	9.2E+00	91	1.3E-11
kT(1v+1h)		3.5E-01	13.0		2.9E+01	7.2E+00	92	9.8E-12
SOLUBILITY	mg/l	2,900	14.0		2.2E+01	5.6E+00	93	7.7E-12
LENGTH	FEET	100	15.0		1.7E+01	4.4E+00	94	6.0E-12
VELOCITY	FT/YR	1.269	16.0		1.4E+01	3.4E+00	95	4.7E-12
PART. COEFF		0.4	17.0		1.1E+01	2.7E+00	96	3.6E-12
(t*)i	YEARS	79	18.0		8.3E+00	2.1E+00	97	2.8E-12
C(RTI)/c(gwti)		1.4E-12	19.0		6.5E+00	1.6E+00	98	2.2E-12
			20.0		5.1E+00	1.3E+00	99	1.7E-12
			22.5		2.7E+00	6.8E-01	101	9.4E-13
			25.0		1.5E+00	3.7E-01	104	5.0E-13
			27.5		8.0E-01	2.0E-01	106	2.7E-13
			30.0		4.3E-01	1.1E-01	109	1.5E-13
			32.5		2.3E-01	5.8E-02	111	7.9E-14
			35.0		1.2E-01	3.1E-02	114	4.3E-14
			37.5		6.7E-02	1.7E-02	116	2.3E-14
			40.0		3.6E-02	9.0E-03	119	1.2E-14
			42.5		1.9E-02	4.9E-03	121	6.7E-15
			45.0		1.1E-02	2.6E-03	124	3.6E-15
			47.5		5.7E-03	1.4E-03	126	1.9E-15
			50.0		3.1E-03	7.6E-04	129	1.0E-15
			52.5		1.6E-03	4.1E-04	131	5.6E-16
			55.0		8.9E-04	2.2E-04	134	3.0E-16
			57.5		4.8E-04	1.2E-04	136	1.6E-16
			60.0		2.6E-04	6.4E-05	139	8.8E-17
			62.5		1.4E-04	3.5E-05	141	4.7E-17
			65.0		7.5E-05	1.9E-05	144	2.6E-17
			67.5		4.0E-05	1.0E-05	146	1.4E-17
			70.0		2.2E-05	5.4E-06	149	7.4E-18

ECC RISK ASSESSMENT
SEPT. 30, 1987

TOLUENE			YEAR	INITIAL CONC. ppb	C(LT) ppb	C(GWT) ppb	YEAR	C(RTI) ppb
CONCENTRATION	ppm	121	0.0	1.2E+05	1.2E+03	3.1E+02	287	3.1E+02
Di		0.07834	1.0		1.2E+03	3.1E+02	288	3.1E+02
p		28	2.0		1.2E+03	3.1E+02	289	3.1E+02
MWi		92.30	3.0		1.2E+03	3.1E+02	290	3.0E+02
Csi	pMWi/62.3*293	1.4E-04	4.0		1.2E+03	3.0E+02	291	3.0E+02
A		10,000	5.0		1.2E+03	3.0E+02	292	3.0E+02
Pt		0.35	6.0		1.2E+03	3.0E+02	293	3.0E+02
Pt^(4/3)		2.5E-01	7.0		1.2E+03	3.0E+02	294	3.0E+02
dsc	30.48*2.5	7.6E+01	8.0		1.2E+03	3.0E+02	295	3.0E+02
k(1v)		5.7E-03	9.0		1.2E+03	3.0E+02	296	3.0E+02
HALF-LIFE	YEARS	NONE	10.0		1.2E+03	3.0E+02	297	3.0E+02
LN(2)		6.9E-01	11.0		1.2E+03	3.0E+02	298	2.9E+02
k(1h)		0.0E+00	12.0		1.2E+03	2.9E+02	299	2.9E+02
kT(1v+1h)		5.7E-03	13.0		1.2E+03	2.9E+02	300	2.9E+02
SOLUBILITY	mg/l	515	14.0		1.2E+03	2.9E+02	301	2.9E+02
LENGTH	FEET	100	15.0		1.2E+03	2.9E+02	302	2.9E+02
VELOCITY	FT/YR	0.348	16.0		1.2E+03	2.9E+02	303	2.9E+02
PART. COEFF		0.055	17.0		1.2E+03	2.9E+02	304	2.9E+02
(t*)i	YEARS	287	18.0		1.2E+03	2.9E+02	305	2.9E+02
C(RTI)/c(gwti)		1.0E+00	19.0		1.1E+03	2.9E+02	306	2.9E+02
			20.0		1.1E+03	2.9E+02	307	2.8E+02
			22.5		1.1E+03	2.8E+02	310	2.8E+02
			25.0		1.1E+03	2.8E+02	312	2.8E+02
			27.5		1.1E+03	2.8E+02	315	2.8E+02
			30.0		1.1E+03	2.7E+02	317	2.7E+02
			32.5		1.1E+03	2.7E+02	320	2.7E+02
			35.0		1.1E+03	2.7E+02	322	2.7E+02
			37.5		1.1E+03	2.7E+02	325	2.7E+02
			40.0		1.1E+03	2.6E+02	327	2.6E+02
			42.5		1.0E+03	2.6E+02	330	2.6E+02
			45.0		1.0E+03	2.6E+02	332	2.6E+02
			47.5		1.0E+03	2.6E+02	335	2.5E+02
			50.0		1.0E+03	2.5E+02	337	2.5E+02
			52.5		1.0E+03	2.5E+02	340	2.5E+02
			55.0		9.9E+02	2.5E+02	342	2.5E+02
			57.5		9.8E+02	2.5E+02	345	2.4E+02
			60.0		9.7E+02	2.4E+02	347	2.4E+02
			62.5		9.6E+02	2.4E+02	350	2.4E+02
			65.0		9.5E+02	2.4E+02	352	2.4E+02
			67.5		9.4E+02	2.4E+02	355	2.3E+02
			70.0		9.3E+02	2.3E+02	357	2.3E+02

ECC RISK ASSESSMENT
SEPT. 30, 1987

ETHYLBENZENE			YEAR	INITIAL CONC. ppb	C(LT) ppb	C(GWT) ppb	YEAR	C(RTI) ppb
CONCENTRATION	ppm	69.1	0.0	6.9E+04	5.7E+02	1.4E+02	575	1.4E+02
Di		0.06672	1.0		5.7E+02	1.4E+02	576	1.4E+02
p		7	2.0		5.7E+02	1.4E+02	577	1.4E+02
MWi		106.17	3.0		5.7E+02	1.4E+02	578	1.4E+02
Csi	pMWi/62.3*293	4.1E-05	4.0		5.7E+02	1.4E+02	579	1.4E+02
A		10,000	5.0		5.7E+02	1.4E+02	580	1.4E+02
Pt		0.35	6.0		5.7E+02	1.4E+02	581	1.4E+02
Pt^(4/3)		2.5E-01	7.0		5.7E+02	1.4E+02	582	1.4E+02
dsc	30.48*2.5	7.6E+01	8.0		5.7E+02	1.4E+02	583	1.4E+02
k(1v)		1.4E-03	9.0		5.6E+02	1.4E+02	584	1.4E+02
HALF-LIFE	YEARS	NONE	10.0		5.6E+02	1.4E+02	585	1.4E+02
LN(2)		6.9E-01	11.0		5.6E+02	1.4E+02	586	1.4E+02
k(1h)		0.0E+00	12.0		5.6E+02	1.4E+02	587	1.4E+02
kT(1v+1h)		1.4E-03	13.0		5.6E+02	1.4E+02	588	1.4E+02
SOLUBILITY	mg/l	152	14.0		5.6E+02	1.4E+02	589	1.4E+02
LENGTH	FEET	100	15.0		5.6E+02	1.4E+02	590	1.4E+02
VELOCITY	FT/YR	0.174	16.0		5.6E+02	1.4E+02	591	1.4E+02
PART. COEFF		0.06	17.0		5.6E+02	1.4E+02	592	1.4E+02
(t*)i	YEARS	575	18.0		5.6E+02	1.4E+02	593	1.4E+02
C(RTI)/c(gwti)		9.9E-01	19.0		5.6E+02	1.4E+02	594	1.4E+02
			20.0		5.6E+02	1.4E+02	595	1.4E+02
			22.5		5.6E+02	1.4E+02	597	1.4E+02
			25.0		5.6E+02	1.4E+02	600	1.4E+02
			27.5		5.5E+02	1.4E+02	602	1.4E+02
			30.0		5.5E+02	1.4E+02	605	1.4E+02
			32.5		5.5E+02	1.4E+02	607	1.4E+02
			35.0		5.5E+02	1.4E+02	610	1.4E+02
			37.5		5.5E+02	1.4E+02	612	1.4E+02
			40.0		5.5E+02	1.4E+02	615	1.4E+02
			42.5		5.5E+02	1.4E+02	617	1.4E+02
			45.0		5.5E+02	1.4E+02	620	1.4E+02
			47.5		5.4E+02	1.4E+02	622	1.3E+02
			50.0		5.4E+02	1.4E+02	625	1.3E+02
			52.5		5.4E+02	1.4E+02	627	1.3E+02
			55.0		5.4E+02	1.3E+02	630	1.3E+02
			57.5		5.4E+02	1.3E+02	632	1.3E+02
			60.0		5.4E+02	1.3E+02	635	1.3E+02
			62.5		5.4E+02	1.3E+02	637	1.3E+02
			65.0		5.3E+02	1.3E+02	640	1.3E+02
			67.5		5.3E+02	1.3E+02	642	1.3E+02
			70.0		5.3E+02	1.3E+02	645	1.3E+02

ECC RISK ASSESSMENT
SEPT. 30, 1987

PCB AROCLOR 1260			YEAR	INITIAL CONC. ppb	C(LT) ppb	C(GWT) ppb	YEAR	C(RTI) ppb
CONCENTRATION	ppm	1.40	0.0	1.4E+03	3.4E+00	8.6E-01	11,111	8.5E-01
Di		0.0526	1.0		3.4E+00	8.6E-01	11,112	8.5E-01
p		4.5E-05	2.0		3.4E+00	8.6E-01	11,113	8.5E-01
MWi		378.7	3.0		3.4E+00	8.6E-01	11,114	8.5E-01
Csi	pMWi/62.3*293	9.3E-10	4.0		3.4E+00	8.6E-01	11,115	8.5E-01
A	cm^2	10,000	5.0		3.4E+00	8.6E-01	11,116	8.5E-01
Pt		0.35	6.0		3.4E+00	8.6E-01	11,117	8.5E-01
Pt^(4/3)		2.5E-01	7.0		3.4E+00	8.6E-01	11,118	8.5E-01
dsc	30.48*2.5	7.6E+01	8.0		3.4E+00	8.6E-01	11,119	8.5E-01
k(1v)		2.5E-08	9.0		3.4E+00	8.6E-01	11,120	8.5E-01
HALF-LIFE	YEARS	NONE	10.0		3.4E+00	8.6E-01	11,121	8.5E-01
LN(2)		6.9E-01	11.0		3.4E+00	8.6E-01	11,122	8.5E-01
k(1h)		0.0E+00	12.0		3.4E+00	8.6E-01	11,123	8.5E-01
kT(1v+1h)		2.5E-08	13.0		3.4E+00	8.6E-01	11,124	8.5E-01
SOLUBILITY	mg/l	0.08	14.0		3.4E+00	8.6E-01	11,125	8.5E-01
LENGTH	FEET	100	15.0		3.4E+00	8.6E-01	11,126	8.5E-01
VELOCITY	FT/YR	0.009	16.0		3.4E+00	8.6E-01	11,127	8.5E-01
PART. COEFF		0.005	17.0		3.4E+00	8.6E-01	11,128	8.5E-01
(t*)i	YEARS	11,111	18.0		3.4E+00	8.6E-01	11,129	8.5E-01
C(RTI)/c(gwti)		9.9E-01	19.0		3.4E+00	8.6E-01	11,130	8.5E-01
			20.0		3.4E+00	8.6E-01	11,131	8.5E-01
			22.5		3.4E+00	8.6E-01	11,134	8.5E-01
			25.0		3.4E+00	8.6E-01	11,136	8.5E-01
			27.5		3.4E+00	8.6E-01	11,139	8.5E-01
			30.0		3.4E+00	8.6E-01	11,141	8.5E-01
			32.5		3.4E+00	8.6E-01	11,144	8.5E-01
			35.0		3.4E+00	8.6E-01	11,146	8.5E-01
			37.5		3.4E+00	8.6E-01	11,149	8.5E-01
			40.0		3.4E+00	8.6E-01	11,151	8.5E-01
			42.5		3.4E+00	8.6E-01	11,154	8.5E-01
			45.0		3.4E+00	8.6E-01	11,156	8.5E-01
			47.5		3.4E+00	8.6E-01	11,159	8.5E-01
			50.0		3.4E+00	8.6E-01	11,161	8.5E-01
			52.5		3.4E+00	8.6E-01	11,164	8.5E-01
			55.0		3.4E+00	8.6E-01	11,166	8.5E-01
			57.5		3.4E+00	8.6E-01	11,169	8.5E-01
			60.0		3.4E+00	8.6E-01	11,171	8.5E-01
			62.5		3.4E+00	8.6E-01	11,174	8.5E-01
			65.0		3.4E+00	8.6E-01	11,176	8.5E-01
			67.5		3.4E+00	8.6E-01	11,179	8.5E-01
			70.0		3.4E+00	8.6E-01	11,181	8.5E-01

APPENDIX C

ENVIRONMENTAL FATE AND TRANSPORT DESCRIPTION FOR INDICATOR CHEMICALS

APPENDIX C

ENVIRONMENTAL FATE AND TRANSPORT OF THE INDICATOR CHEMICALS FOR THE ENVIRONMENTAL CONSERVATION AND CHEMICAL CORPORATION SITE INDIANA

APPENDIX C

C.1 Chloroform

Chloroform is ubiquitous to the environment, both in urban and non-urban areas. Ninety percent of chloroform's use is in the production of chlorodifluoromethane with minor uses as a solvent, cleaning agent and fumigant ingredient. Chloroform, a dense, colorless, volatile liquid, is the most well-known of the trihalomethanes. From its water solubility and density, any chloroform in excess of its water solubility would sink to he bottom of a water, i.e. "sinker".

The major environmental fate process is volatilization of chloroform from both soil and water to the atmosphere. The volatilization half-life of chloroform in water at 25°C has been calculated at 21 minutes and the overall half life in water estimated at 0.3 to 30 days. Due to the high vapor pressure of chloroform, volatilization into the atmosphere is quite rapid. Once in the troposphere chloroform is attacked by hydroxyl radicals and form CCl_3 radicals which react with oxygen to yield phosgene ($COCl_2$) and possibly chlorine oxide (ClO) radicals. These compounds further hydrolize to HCl and CO_2. Therefore, the primary fate process for chloroform once it has reached the troposphere is oxidation. Studies on adsorption, bioaccumulation, biotransformation/biodegradation of chloroform in the environment are limited in scope and therefore, these fate processes are considered of minor environmental significance. The log octanol/water partition coefficient of chloroform indicates a possible tendency of this compound to bioaccumulate under conditions of constant exposure. However, there is no evidence for biomagnification of chloroform in the aquatic food chain. The potential for biodegradation of chloroform in the aquatic environment was examined and no aerobic biodegradation was observed. However, studies conducted under anaerobic conditions such as in lake sediments for ground water and studies conducted in the presence of methanogenic bacteria, reported the reduction and degradation of chloroform under these conditions. The quantity of information on biodegradation of chloroform in the environment is limited and thus, biodegradation is considered a minor environmental fate process.

The major transport process for chloroform is volatilization from water and soils to the atmosphere with subsequent oxidation in the troposphere.

References: Callahan, M.A., et al, 1979; Mills, W.B., et al, 1982; Mabey, W.R., et al, 1982; EPA, 1985; EPA, 1985g.

C.2 1,1,2-Trichloroethane

1,1,2-Trichloroethane is a colorless liquid that is used in the manufacture of 1,1-dichloroethene and as a solvent for chlorinated rubber and other organic materials. 1,1,2-Trichloroethane can enter both the water and air environments through general use of chlorinated rubber and through lab use. 1,1,2-Trichloroethane is moderately soluble, and will be a "sinker" when its water solubility is exceeded.

A relatively small amount of information is available regarding the important fate processes for 1,1,2-trichloroethane. The fate of this compound in the environment can be inferred from information available on its structural isomer, 1,1,1-trichloroethane. Volatilization is the important transport process for 1,1,2-trichloroethane in aquatic environments. Once in the troposphere, it is believed that photooxidation takes place. Photolysis, hydroloysis, and sorption are not significant environmental fate processes for 1,1,2-trichloroethane. Based on its octanol/water partition coefficient, 1,1,2-trichloroethane will probably not bioaccumulate to any significant extent. Biodegradation of 1,1,2-trichloroethane in the environment is not an important fate process. Similar to other low molecular weight chloroaliphatics, biodegradation is slow.

The important environmental transport process for 1,1,2-trichloroethane is volatilization from water or soils to the atmosphere.

References: Mabey, W.R., et al, 1982; Callahan, M.A., et al, 1979; Verschueren, K., 1983.

C.3 1,1,1-Trichloroethane

1,1,1-Trichloroethane, a colorless liquid with a sweet odor, has uses as a degreaser and solvent. Otherwise include, aerosol formation and in coatings and paints. A moderately water soluble and volatile saturated aliphatic hydrocarbon, 1,1,1-trichloroethane will be a "sinker" if its water solubility is exceeded.

Volatilization is the only important environmental fate process for 1,1,1-trichloroethane. The tropospheric half-life of 1,1,1-trichloroethane is approximately 1 year compared to the statospheric half-life of 2-11 years. The laboratory half-life in surface water is 0.14-7 days. Photolysis, hydrolysis, oxidation, and sorption are not environmentally significant fate processes of 1,1,1-trichloroethane. Information on biodegradation/biotransformation and bioaccumulation s limited and will not be considered as significant fate processes.

The major environmental transport process for 1,1,1-trichloroethane is volatilization from water or soils to the atmosphere.

References: Mabey, W.R., et al, 1982; Callahan, M.A., et al, 1979; Verschueren, K., 1983; Weast R.C., 1974-1975; EPA, 1984a.

C.4 Trichloroethene

Trichloroethene (TCE) is ubiquitous in the environment, although it is not naturally occurring. Widely used as a solvent in industrial degreasing of metals, TCE has minor uses in fumigant mixtures, inhalation anesthesia, and decaffenation of coffee. TCE is a highly volatile unsaturated aliphatic hydrocarbon with a relatively high water solubility. From its density, any TCE in excess of its water solubility would sink to the bottom of the water, i.e., "sinker".

Volatilization of TCE in the environment is the most important fate process with a laboratory half-life of 21 minutes. Once the compound enters the troposphere, high temperatures and UV radiation promote rapid degradation ($t_{1/2}$ = 4 days) to HCl, dichloroacetyl chloride, phosgene, carbon monoxide, and hexachlorobutadiene. The overall half-life of TCE in surface water and air is 1-90 days and 4 days, respectively. Limited laboratory studies on the sorption of TCE onto soils and sediments indicate that TCE does not sorb to a great extent to pure clays (<5 percent sorption). Thus sorption will not be considered as a major fate process. TCE does not significantly bioaccumulate in the environment as seen by bioconcentration factors of 10^{-17} for bluegills with a half-life in tissue of less than 1 day. Biodegradation/biotransformation is of minor significance as an environmental fate process, however, higher mammals, including man, can degrade TCE to chlorinated acetic acids.

Environmental transport of TCE is due solely to volatilization from water to the atmosphere.

References: Callahan, M.A. et al, 1979; Mills, W.B. et al, 1982, EPA 1985b; Schuller, T.A., 1983.

C.5 Tetrachloroethene

Tetrachloroethene (PCE) is a colorless liquid with an etheric odor. It has many uses in dry cleaning operations, metal degreasing, as a solvent, and removing soot from boilers. PCE is also used to manufacture paint removers, ink, trichloroacetic acid, heat transfer medium, and fluorocarbons. PCE is only slightly soluble in water, and when its solubility is exceeded it will be a "sinker".

Volatilization is the major transport process of PCE from the aquatic environment. The average half-life range for PCE volatilizing from aquatic systems has been reported as 20.2 to 27.1 minutes. Once in the troposhere, PCE reacts with hydroxyl radicals resulting in formation of trichloroacetyl chloride as a major product, and phosgene as a minor product. The tropospheric lifetime of PCE is reported to be 10 days. Photolysis and hydrolysis do not seem to be significant environmental fate processes for PCE. Based on its octanol/water partition coefficient, this compound may have the potential to bioaccumulate. As with other chlorinated aliphatics, PCE probably biodegrades in the environment, however, at slow rates. Thus, biodegradation does not seem to be an important fate process.

Volatilization into the troposphere with subsequent photooxidation is the important transport process for PCE.

References: Verschueren, K, 1983; Callahan, M.A., et al, 1979. Mills, W. B. et al, 1982; EPA 1985e.

C.6 Ethylbenzene

Ethylbenzene is a colorless liquid. It is used to manufacture styrene and acetophenone. It is also used as a solvent, an asphalt constituent, and as a naphtha constituent. Ethylbenzene can enter the environment through activities of the petroleum refining and the organic chemical industries. Ethylbenzene is only slightly soluble in water and when its solubility is exceeded, it will float to the top of water, i.e. a "floater".

The major environmental fate process for ethylbenzene is volatilization from soils and waters. Once in the atmosphere, ethylbenzene undergoes rapid photochemical reactions with a corresponding half-life of 15 hours. This process dominates all other fate processes. Little or no quantifiable information is available regarding oxidation, hydrolysis and bioaccumulation of ethylbenzene in aquatic systems, thus these are not considered significant fate processes. Ethylbenzene can be used by some bacteria as a sole carbon source, however, the importance of biodegradation as a fate process is improbable.

The important transport process for ethylbenzene is volatilization into the atmosphere and subsequent photooxidation.

References: Verschueren, K., 1983; Callahan, M.A., et al, 1979. Mills, W.B. et al, 1982.

C.7 Toluene

Toluene is a flammable colorless liquid with a sour or burnt odor. It is moderately soluble in water but is miscible with most other organic solvents. Toluene occurs naturally as a component of petroleum oil and is produced indirectly in large volumes during gasoline refining and other operations. The main uses for toluene are as a raw material in the production of benzene and other organic solvents, as a solvent (especially for paints, coatings, gums, oils and resins), and as a gasoline additive to elevate octane ratings. This unsaturated aromatic hydrocarbon will float in water if its water solubility is exceeded, i.e., "floater".

The major environmental fate process for toluene is volatilization with an estimated half-life of 5.18 hours. Photooxidation is the primary atmospheric fate process for toluene with benzaldehyde as the principal organic product reported. Direct photolysis of toluene in the troposphere is energetically improbable while oxidation and hydrolysis in aquatic systems are probably not important. Little quantifiable information was found in the literature concerning the photolysis, hydrolysis, oxidation, biodegradation, and bioaccumulation of toluene in the environment. Therefore, these processes are considered to be of minor environmental significance. The biodegradation potential of toluene indicates that this compound would probably eventually degrade in the environment, but not at a substantial rate.

The major environmental transport process for toluene is volatilization from soils or surface water (or both) to the atmosphere as well as fugitive dust emissions and dry deposition of toluene and oxidation products to the aquatic and terrestrial environments.

References: Callahan, M.A., et al, 1979; Mabey, W.R., et al, 1982; Mills, W.B., et al, 1982; Verschueren, K., 1983; EPA, 1983; EPA, 1985f; EPA, 1985g.

C.8 Phenol

Phenol, a white crystalline substance with a characteristic aromatic acrid odor, has a high water solubility and low vapor pressure. The major uses of phenol are bulk productions of resins and other specialty chemicals, including pharmaceuticals, dyes, and salicylic acid. Phenol is classified as a "sinker" in water based upon its density and water solubility.

Photolysis and biodegradation appear to be major fate processes for phenol in aquatic environments. Photolysis by-products include 4,4-dihydroxybiphenyl and 2,4-dihydroxybiphenyl. Biodegradation of phenols occurs fairly rapidly ($t_{1/2}$ = <1 day) and eventually produces carbon monoxide and water. Overall half-lives have been calculated for phenol in air and surface water as 1-9 days in each case. Oxidation may play a part in phenol's fate in the atmosphere, but sufficient data is lacking. At this time, little data is available for volatilization, sorption, and bioaccumulation of phenol in the environment. However, based upon the physical-chemical properties of phenol (K_{OC}, K_{OW}, etc.), these fate processes do not appear significant.

The major transport processes for phenol in the environment are biodegradation and photolysis.

References: Mabey, W.R. et al, 1982; Callahan, M.A. et al, 1979; Verschueren, K., 1983; EPA 1985c; Mills, W.B. et al, 1982; University of Michigan, 1976.

C.9 Polychlorinated Biphenyls

Polychlorinated biphenyls (PCBs) are a class of chlorinated, aromatic hydrocarbons which had widespread use due to their stability and chemical inertness as well as their dielectric properties. PCBs are widely varied in their physical (oil to liquid to resins) and chemical (soluble to insoluble) properties. In general, PCBs as a class are liquid, denser than water, insoluble in water, and non-volatile. Numerous uses based upon the the properties of PCBs include dielectric fluids, fire retardants, and plasticizers.

Biotransformation/biodegradation are important fate processes for the mono-, di-, and tri-chlorinated biphenyls, intermediate importance for tetrachlorinated biphenyls, and no importance for penta- and higher chlorinated biphenyls which are completely resistant. Lesser chlorinated hydrocarbons are biotransformed in the environment to chlorobenzoic acids and chlorophenylglyoxylic acid. Sorption, volatilization (aerosol distribution followed by fallout with dust or rain and fugitive dust emissions), and bioaccumulation are other important fate processes. PCBs strongly sorb to sediments and/or suspended particles resulting in extremely long half-lives ($t_{1/2}$ = 52.5 days) and making desorption a possibility for years to come. Volatilization of PCBs results from fugitive dust emissions ($t_{1/2}$ = 10.4 hours). PCBs strongly bioaccumulate in the food chain due to the desorption from sediments and direct uptake by plants and other aquatic species. Experiments with Daphnia magna show a tendency for the bioconcentration factor to increase with increasing chlorine content or decreasing water solubility. Photolysis is a minor fate process for PCBs in natural surface waters. PCBs can be partially dechlorinated with shortwave UV light to yield chlorinated biphenylenes and chlorinated dibenzofurans. Photolysis of PCBs requires an oxygen-depleted atmosphere, however, the photic zone in natural waters is oxygen-rich due to photosynthesis and reaeration. PCBs are fairly stable (resistant) to hydrolysis and oxidation.

Environmental transport processes for PCBs include volatilization from soils and surface waters; sediments; adsorption onto soil particles which leads to sedimentation; desorption from soil particles and sediments which leads to re-solution; bioconcentration in the food chain; biodegradation of lesser chlorinated hydrocarbons; fugitive dust emissions which lead to volatilization and precipitation; and to a small extent, photolysis.

References: Mabey et al, 1982; Callahan et al, 1979; Verschueren 1983; Mills et al, 1982; EPA 1985g; Safe, S. 1983; D'Itri, F.M. 1983.

C.10 Bis(2-ethylhexyl)phthalate

Bis(2-ethylhexyl)phthalate (DEHP), a colorless, odorless oily liquid, is used in the manufacture of plasticizers and plastics. DEHP, one of the most commonly discharged priority pollutants, is insoluble in water, but soluble in mineral oils. Based upon its density and water solubility, excess DEHP will be a "sinker".

Sorption, biodegradation, and bioaccumulation of DEHP are competing fate processes in the environment. The predominant fate process depends upon the type of aquatic and soil environments present at a site. The available quantitative sorption data and K_{OC} for DEHP indicated that sorption to soils/sediments is a highly probably fate process. DEHP is a lipophilic compound which bioaccumulates in the aquatic food chain and also in higher mammals. This bioaccumulation is followed by metabolism and excretion, thus, biomagnification in the food chain is not likely. Bioconcentration factors range from 70 to 13,400 times the water concentration. DEHP is readily biodegraded to the corresponding di-carboxylic acid ($t_{1/2}$ approximately 4 weeks). Limited information exists concerning the photolysis, hydrolysis (calculated $t_{1/2}$ = 2,000 years), oxidation, and volatilization of DEHP in the environment.

Environmental transport processes for DEHP include sorption to soils, sediments, and/or suspended particles, biodegradation, and bioaccumulation.

References: Callahan, M.A., et al, 1979, Mabey, W.R., et al, 1982; Mills, W.B., et al, 1982; Perwak, J. et al 1981.

C.11 Di-n-butyl phthalate

Di-n-butyl phthalate (DBP), a colorless, odorless oily liquid, is used in the manufacture of plasticizers and plastics. Minor uses include cosmetics, industrial stains manufacture, safety glass manufacture, insecticides, printing inks, paper coatings, and adhesives. Man-caused sources include the general use of plastics, microcontamination and laboratory chemicals, food, detergents, and also from lipsticks, paints, and insecticides. DBP is insoluble in water, but soluble in mineral oils. Based upon its density and water solubility, excess DBP will be a "sinker".

Sorption, biodegradation, and bioaccumulation of DBP are competing fate processes in the environment. The predominant fate process depends upon the type of aquatic and soil environment present at a site. The K_{OC} and the available quantitative sorption data for DBP indicate that sorption to soils/sediments is a highly probable fate process. DBP is a lipophilic compound which bioaccumulates in a variety of aquatic organisms. This bioaccumulation is followed by metabolism and excretion, thus, biomagnification in the food chain is not likely. DBP is degraded under most conditions and can be metabolized by multicellular organisms. Limited information exists concerning the photolysis, oxidation, volatilization, and hydrolysis (calculated $t_{1/2}$ = 3.2 years) of DBP in the environment.

Environmental transport processes for DBP include sorption to soils, sediments, and/or suspended particles, biodegradation, and bioaccumulation. Sorption onto suspended particles and biota is probably the more important transport mechanism in the environment.

References: Mabey, W.R. et al 1982; Callahan, M.A., et al, 1979; Mills, W.B., et al, 1982; Perwak, J. et al, 1981.

C.12 Diethyl Phthalate

Diethyl phthalate (DEP) is an odorless liquid that has many common uses. It has uses in plastic manufacturing and processing, food packaging materials, insecticides and insect repellants, and as a dye application agent. DEP is not very soluble in water and when it exceeds its solubility, it tends to sink to the bottom of the water, i.e. a "sinker".

Not very much is known about DEP in the environment. Not much data is available in the literature, thus photolysis, oxidation, and volatilization are considered minor fate processes. Hydrolysis is considered to take place in aquatic environments, however, it is not considered a competitive process with a half-life reported to be about 18.3 years. Sorption and complexation with humic substances is probably an important transport process. Based on its octanol/water partition coefficient and behavior of other phthalate esters, bioaccumulation would probably occur. Studies indicate that metabolism and excretion does occur, thus biomagnification in the food-chain is unlikely. Similarly, biodegradation also occurs, and is considered to probably be an important fate process.

Environmental fate and transport processes important to DEP include sorption to particulates, complexation with humic substances, bioaccumulation, and biodegradation.

References: Verschueren, K, 1983; Callahan, M.A., et al, 1979; Mabey, W.R., et al, 1982; Mills, M.B., et al, 1982; Perwak, J et al, 1981.

C.13 Dimethyl phthalate

Dimethyl phthalate (DMP) is a colorless liquid that is relatively odorless. It has various uses in the plastics and rubber industries. DMP is the most water soluble of the phthalate esters and is considered only moderately soluble in water. When its solubility in water is exceeded, it will be a "sinker".

Like most of the phthalate esters, little is known about the fate and transport of DMP in the environment. Photolysis, oxidation and volatilization are not considered significant environmental fate processes for DMP. Hydrolysis of DMP does occur but at slow rates (half-life=3.2 years,) and thus is not considered significant. Sorption to suspended particles, soils, and humic substances is considered the most important transport mechanisum for DMP. Bioaccumulation and biodegradation of DMP does occur. Based on its octanol/water partition coefficient, DMP is lipophilic. Both bioaccumulation and biodegradation are considered important fate processes.

The major transport process for DMP in the environment is sorption to soil and/or suspend particles and humics. The important fate processes are bioaccumultion and biodegradation.

References: Verschueren, K., 1983; Callahan, M.A., et al, 1979; Perwak, J. et al, 1981.

APPENDIX D

TOXICOLOGICAL PROFILES OF THE INDICATOR CHEMICALS

APPENDIX D

TOXICOLOGY PROFILES OF THE INDICATOR CHEMICALS FOR THE ENVIRONMENTAL CONSERVATION AND CHEMICAL CORPORATION SITE INDIANA

APPENDIX D

D.1 Chloroform (EPA 1985, 1985g; MacKison et al, 1981;

D.1.1 Summary of Health Effects Data

Chloroform (trichloromethane) is produced during the chlorination of drinking water and thus is a common drinking water contaminant. Chronic administration of chloroform by gavage is reported to produce a dose-related increase in the incidence of kidney epithelial tumors in rats and a dose-related increase in the incidence hepatocellular carcinomas in mice. Epidemiological studies suggest that higher concentrations of chloroform and other trihalomethanes in water supplies may be associated with the increased frequency of bladder cancer in humans. However, these results are not sufficient to establish causality. An increased incidence of fetal abnormalities in the offspring of pregnant rats exposed to chloroform by inhalation has been observed. Oral doses of chloroform that cause maternal toxicity produce relatively mild fetal toxicity in the form of reduced birth weight. There are limited data suggesting that chloroform has mutagenic activity in some test systems. However, negative results have been reported for bacterial mutagenesis assays.

Humans may be exposed to chloroform by inhalation, ingestion, or skin contact. Toxic effects include local irritation of the skin or eyes, central nervous system depression, gastrointestinal irritation, liver and kidney damage, cardiac arrhythmia, ventricular tachycardia, and bradycardia. Death from chloroform overdose can occur and is attributed to ventricular fibrillation. Chloroform anesthesia can produce delayed death as a result of liver necrosis. Exposure to chloroform by inhalation, intragastric administration, or intraperitoneal injection produced liver and kidney damage in laboratory animals. The oral LD_{50} and inhalation LC_{LO} values for the rat are 908 mg/kg and 39,000 mg/m^3 for four hours, respectively.

D.1.2 Pharmacokinetics and Metabolism

The absorption routes of chloroform into the body are dermal, inhalation and oral. Dermal absorption of chloroform vapors is negligible; however, direct contact with pure liquids permits a slow absorption process. Due to chloroform's high vapor pressure, inhalation is normally the principal route of entry into the body. The total amount absorbed via the lungs is directly proportional to 1) the concentration of the inspired air, 2) the duration and time of exposure, 3) the blood/air Ostwald solubility co-efficient, 4) the solubility in the various body tissues, and 5) physical activity. Once in the body, chloroform is biotransformed and the metabolites excreted either

in expired air or through the urine. Total elimination of absorbed unchanged chloroform is through expired air.

D.1.3 Toxic and Carcinogenic Studies

Chloroform is among the morethan fifty chemicals evaluated by CAG for relative carcinogenic potencies as suspected human carcinogens. A level-of-evidence in animals indicates that sufficient studies have been conducted to determine the carcinogenicity of chloroform. However, inadequate studies have been conducted to determine the level of carcinogenic evidence in humans. Therefore, IARC has ranked chloroform as a B2 ("probable" human carcinogen) compound based upon the level of evidence in animal studies.

D.1.4 Applicable and Relevant Standards

The recognized applicable and relevant standards for chloroform are summarized in Table D-1. The ambient water quality criterion for the protection of fresh water life is <28.9 mg/L. A MCL has been established at 0.1 mg/L for trihalomethanes (chloroform). Regulations for workplace exposure are 50 ppm (240 mg/m^3) for OSHA and 10 ppm (50 mg/m^3) for ACGIH.

D.2 1,1,2-Trichloroethane (EPA 1981, 1984a, 1985f, 1986g)

D.2.1 Summary of Health Effects Data

1,1,2-Trichloroethane depresses the central nervous system causing narcosis, in which respect it is considerably more potent than chloroform. By inhalation its acute toxicity is somewhat greater for certain laboratory animals (cats) than that of chloroform. Narcotic concentrations of 1,1,2-trichloroethane result in irritation to the eyes and nose and injection of the conjunctiva. Death occurs from respiratory arrest. Concentrations producing deep narcosis and death are of the order of 13,600 ppm for a two-hour exposure. The corresponding concentration for chloroform is 30,000 to 40,000 ppm. 1,1,2-Trichloroethane is lethal by oral and subcutaneous administration; 0.75 g/kg was lethal to dogs by mouth, compared with 2.25 g/kg for chloroform. Fatty degeneration of the liver was observed in dogs dying two or more days following administration of 1,1,2-trichloroethane, which is also absorbed through the intact skin.

More recent data include the following oral LD_{50}, rat-580 mg/kg; intraperitoneal LD_{50}, mouse-494 mg/kg, dog-450 mg/kg; subcutaneous LD_{40} mouse-227 mg/kg. Exposure at 500 ppm for 8 hours was fatal to rats.

TABLE D-1.

Summary of Toxicological Information for Chloroform

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA MCL (trihalomethanes, mg/L)	0.1
EPA Water Quality Criteria (mg/L)	
fish and drinking water	1.90E-07
fish only	1.57E-02
protection of aquatic life	<28.9
EPA Drinking Water Health Advisories (mg/L)	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA ppm(mg/m3)	50 (240)
ACGIH 8 hr TWA ppm(mg/m3)	10 (50)
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	
AIC	none
AIS	none
ADI	none
inhalation (mg/kg/day)	
AIC	none
AIS	none
ADI	none
median effective dose (mg/day)	
oral	none
inhalation	none
Carcinogenic effects	
Carcinogenic Potency Factor (1.0E-06 cancer risk)	
oral 1/(mg/kg/day)	8.10E-02
inhalation	none
10% effective dose(mg/kg/day)	
oral	0.508
inhalation	0.508
Cancer Risk	
Inhalation at 1 ug/m3 (risk)	2.30E-06
water (ug/L,1.0E-06 risk)	1.90E-04
Classification, EPA	B2
Classification, IARC	2B

D.2.2 Pharmacokinetics and Metabolism

1,1,2-Trichloroethane can affect the body if it is inhaled, comes in contact with the eyes or skin, or is ingested. It may also be absorbed through the skin. An intraperitioneal dose of 1,1,2-trichloroethane in mice resulted in expiration of 16-20% and urinary excretion of 73-87% of the original dose. In mice, the major urinary metbolites are S-carboxymethyl cysteine, chloroacetic acid, and thiodiacetic acid. Minor metabolites included oxalic acid, 2,2-dichloroethanol, glycolic acid, and trace amounts of 2,2,2-trichloroethanol and trichloroeacetic acid. These metabolites suggest a metabolic pathway via formation of chloroacetaldehyde. Only 1-3% of the original dose remained in the animal after 3 days with 0.1-2% in the feces.

D.2.3 Toxic and Carcinogenic Studies

1,1,2-Trichlorethane vapor is a potent narcotic. Injury to lungs, liver, and kidneys has been observed in animals. The lethal concentration for rats was 2000 ppm for 4 hours. Concentrations resulting in narcosis also caused irritation of the nose and eyes. Mice treated by intraperitoneal injection with anesthetic doses showed moderate hepatic dysfunction and renal dysfunction. At autopsy, there was centrolobular necrosis of the liver and tubular necrosis of the kidney. No human cases of intoxication or systemic effects from industrial exposure have been reported.

D.2.4 Applicable Standards

The recognized applicable and relevant standards for 1,1,2-trichloroethane are summarized in Table D-2. The ambient water quality criterion for the protection of fresh water life is <18.0 mg/L. 1,1,2-trichloroethane has been established at 5.73×10^{-2} (mg/kg/day). Time-weighted average (TWA) for work place exposures have been established at 10 ppm or 45 mg/m^3 by OSHA and ACGIH. 1,1,2-trichloroethane is considered a Class C carcinogen by EPA.

D.3 1,1,1-Trichloroethane (EPA 1981, 1984a, 1985f, 1986g

D.3.1 Summary of Health Effects Data

Preliminary results suggest that 1,1,1-trichloroethane induces liver tumors in female mice. It has been shown to be mutagenic in the Ames Assay and it causes transformation in cultured rat embryo cells. Inhalation exposure to high concentrations of 1,1,1-trichloroethane depresses the central nervous system; affects cardiovascular functions; and damages the lungs, liver, and kidneys in animals and humans. Irritation of the skin and mucous membranes has also been associated with human exposure to 1,1,1-trichloroethane (350 ppm and above). The oral LD_{50} value

TABLE D-2.

Summary of Toxicological Information for 1,1,2-Trichloroethane

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA MCL (ug/L)	none
EPA Water Quality Criteria (mg/L)	
fish and drinking water	6.04E-04
fish only	4.18E-02
protection of aquatic life	<18.0
EPA Drinking Water Health Advisories (mg/L)	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA(mg/m3)	45
ACGIH 8 hr TWA(mg/m3)	45
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	
AIC	none
AIS	none
ADI	none
inhalation (mg/kg/day)	
AIC	none
AIS	none
ADI	none
median effective dose (mg/day)	
oral	none
inhalation	none
Carcinogenic effects	
Carcinogenic Potency Factor (1.0E-06 cancer risk)	
oral 1/(mg/kg/day)	5.73E-02
inhalation	none
10% effective dose(mg/kg/day)	
oral	2.78E+00
inhalation	2.78E+00
Cancer Risk	
Inhalation at 1 ug/m3 (risk)	
water (ug/L,1.0E-06 risk)	
Classification, EPA	6.00E-04
Classification, IARC	C

for 1,1,1-trichloroethane in rats is about 11,000 mg/kg. The acute toxicity of 1,1,1-trichloroethane to aquatic species is rather low, with the LD_{50} concentrations for the most sensitive species tested being 52.8 mg/L. No chronic toxicity studies have been conducted on 1,1,1-trichloroethane, but acute-chronic ratios for the other chlorinated ethanes range from 2.8-8.7.

D.3.2 Pharmacokinetics and Metabolism

1,1,1-Trichloroethane is rapidly absorbed through the lungs, gastrointestinal tract, and somewhat slower through skin in both man and rodents. Direct contact with pure liquid permits appreciable absorption by the latter route evidenced by the presence of 1,1,1-trichloroethane in the expired air of dermally exposed human volunteers. Distribution occurs throughout the body in all tissues and organs with highest concentrations found in liver followed by brain, kidney, muscle, lung and blood. In controlled human studies, approximately 4% of the total uptake of 1,1,1-trichloroethane is metabolized and excreted in the urine mainly as trichloroethanol and trichloroacetic acid. The remaining absorbed dose is rapidly eliminated unchanged via the lungs.

D.3.3 Toxic and Carcinogenic Studies

1,1,1-Trichloroethane has a low acute toxicity in mammals. In high concentrations this substance may cause CNS depression and has been demonstrated to possess marked narcotic effects in man. Liver and kidney damage has been reported at very high exposures and the hepatotoxic action has been verified in animal experiments, an effect which is potentiated by simultaneous exposure to compounds like acetone or isopropanol (Plaa, G.L. et al in Alcoholic Liver Pathology (Khanna, J.M. et al Edts.) Addition Research Foundation, Toronto 1975, pp 225-244). Limited quantitative data are available concerning long-term exposure to 1,1,1-trichloroethane, mainly deriving from three carcinogenicity studies. On basis of findings of reduced survival EPA has derived an ADI of 0.54 mg/kg/day using an uncertainty factor of 1000. However, since the reduced survival was mostly due to chronic murine pneumonia in the experimental animals, this ADI is of questionable value.

Poor survival or inadequate exposure has rendered the three carcinogenicity studies inadequate in determining carcinogenic risks. Limited data suggest no teratogenic effects in mice or rats, and mutagenic data are also inadequate.

D.3.4 Applicable Standards

The recognized applicable and relevant standards for 1,1,1-trichloroethane are summarized in Table D-3. The ambient water quality criterion for the protection of fresh water life is <18.0 mg/L. A maximum concentration level in drinking water

TABLE D-3.

Summary of Toxicological Information for 1,1,1-Trichloroethane

Relevant Requirements, Criteria, Advisories or Guidance	Value	
EPA MCL (ug/L)	0.2	
EPA Water Quality Criteria (mg/L)		
fish and drinking water	0.0184	
fish only	1.03	
protection of aquatic life	<18.0	
EPA Drinking Water Health Advisories (mg/L)	10 kg	70 kg
1 day	140	---
10 days	35	---
chronic	35	12.5
OSHA 8 hr TWA(mg/m3)	1900 (350 ppm)	
ACGIH 8 hr TWA(mg/m3)	1900 (350 ppm)	
Noncarcinogenic effects		
risk characterization		
oral (mg/kg/day)		
AIC	3.00E-01	
AIS		
inhalation (mg/kg/day)	4.30E-01	
AIC	1.50E+00	
AIS	6.30E+00	
median effective dose (mg/day)		
oral	5.45E+03	
inhalation	5.45E+03	
Carcinogenic effects		
Carcinogenic Potency Factor (1.0E-06 cancer risk)		
oral	none	
inhalation	none	
10% effective dose		
oral	none	
inhalation	none	
Cancer Risk		
Inhalation at 1 ug/m3 (risk)	3.00E-09	
water (ug/L,1.0E-06 risk)	21.7	
Classification, EPA	noncarcinogen	
Classification, IARC	no ranking	

(MCL) has been established at 0.2 mg/L for 1,1,1-trichloroethane. A tentative acceptable daily intake (ADI) for the noncarcinogenic effects of 1,1,1-trichloroethane has been established at 0.54 mg/kg/day. Time-weighted average (TWA) for work place exposures have been established at 350 ppm or 1900 mg/m^3 by OSHA and ACGIH. As previously noted, 1,1,1-trichloroethane is considered a noncarcinogen according to EPA.

D.4 Trichloroethene (EPA 1985f, 1985g; Mackison et al, 1981)

D.4.1 Summary of Health Effects Data

Trichloroethene (TCE) has a low acute toxicity with an acute oral LD_{50} value in several species ranging from 6000-7000 mg/kg. Chronic exposure in rodents have been found to cause adverse effects on liver and kidneys at high doses. In long-term studies TCE has induced hepatocellular carcinomas in mice. Due to presence of carcinogenic impurities in the test compounds and other factors, the significance of these findings are not clear. Extensive epidemiological investigations have failed to substantiate an increased carcinogenic risk in man. Also, results from short-term testing have been ambiguous.

D.4.2 Pharmacokinetics and Metabolism

Trichloroethene can be absorbed by dermal or oral contact or by inhalation. Direct contact with the pure liquid which will permit some absorption which normally is not sufficiently high as to elicit toxic effects. Upon ingestion trichloroethene is readily absorbed, but inhalation usually represents the major route of absorption. Pulmonary uptake of the substance is rapid and distribution occurs to all body tissues with a considerable fraction in adipose tissue. An appreciable part of the TCE absorbed is rapidly excreted unchanged in exhaled air. But the substance is also extensively metabolized (in man 40-70% of the retained dose) mainly by the liver into exhaled carbon dioxide (minor metabolite) as well as into the urinary metabolites trichloroethanol, trichloroacetic acid (major metabolite), and a glucoronide conjugate of trichloroethanol. Although elimination from fatty tissues occur at a slower rate, virtually all TCE is excreted within 48 hours after administration of a single high dose of TCE.

D.4.3 Toxic and Carcinogenic Studies

TCE has a low acute toxicity in mammals. In man higher concentrations of this volatile substance has anesthetic and analgesic properties and is known to occasionally elicit cardiac arrythmias. Chronic exposure has been reported to induce neurotoxic symptoms like ataxia, sleep disturbances and psychotic episodes as well as trigeminal neuropathy.

In rodents TCE causes toxic effects to the kidney tubuli and liver. No significant signs of developmental toxicity has been found in inhalation experiments using these experimental animals.

D.4.4 Applicable and Relevant Standards

The recognized applicable and relevant standards for TCE are summarized in Table D-4. The ambient water quality criterion for the protection of freshwater life is <45.0 mg/L. An MCL in drinking water has been established at 0.005 mg/L for TCE. Regulations for workplace exposures have been developed by OSHA (100 ppm TWA or 540 mg/m^3) and ACGIH (50 ppm or 270 mg/m^3). EPA has classified TCE as a Group B2 carcinogen.

D.5 Tetrachloroethene (Mackison et al, 1981; EPA 1985f, 1985e, 1986b)

D.5.1 Summary of Health Effects Data

Tetrachloroethene (PCE) was found to produce liver cancer in male and female mice when administered orally by gavage. Unpublished gavage studies in rats and mice performed by the National Toxicology Program (NTP) showed hepatocellular carcinomas in mice and a slight, statistically insignificant increase in a rare type of kidney tumor. NTP is also conducting an inhalation carcinogenicity study. Elevated mutagenic activity was found in Salmonella strains treated with tetrachloroethene. Delayed ossification of skull bones and sternebrae were reported in offspring of pregnant mice exposed to 2,000 mg/m^3 of tetrachloroethene for 7 hours/day on days 6-15 of gestation. Increased fetal resorptions were observed from exposure of pregnant rats to tetrachloroethene. Renal toxicity and hepatotoxicity have been noted following chronic inhalation exposure of rate to PCE levels of 1,356 mg/m^3. During the first 2 weeks of a subchronic inhalation study, exposure to concentrations of 1,622 ppm (10,867 mg/m^3) of PCE produced signs of central nervous system depression, and cholinergic stimulation was observed among rabbits, monkeys, rats, and guinea pigs.

D.5.2 Pharmacokinetics and Metabolism

Tetrachloroethene (PCE) can be absorbed into the body by dermal or oral contact or by inhalation. Single oral doses of PCE were absorbed completely when administered to rats (180 mg/kg) and mice (500 mg/kg). Human volunteers at rest absorbed about 25 percent of PCE administered via inhalation (72 to 144 ppm over a four-hour exposure). The compound initially was absorbed rapidly with decreasing uptake as exposure continued. Once in the bloodstream, PCE tends to distribute to body fat. PCE levels in rats rise continuously with the duration of exposure in brain, lungs, and fat but tend to level off in the blood and liver after a 3-hour exposure. Humans metabolize less than 4 percent of the original dose. The proposed metabolic pathway of PCE is

TABLE D-4.

Summary of Toxicological Information for Trichloroethene

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA MCL (mg/L)	0.005
EPA MCLG (proposed)	0
EPA Water Quality Criteria (mg/L)	
fish and drinking water	2.70E-04
fish only	8.07E-02
protection of aquatic life	45.0
EPA Drinking Water Health Advisories (mg/L)	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA ppm(mg/m3)	100 (540)
ACGIH 8 hr TWA ppm(mg/m3)	50 (270)
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	
AIC	none
AIS	none
ADI	none
inhalation (mg/kg/day)	
AIC	none
AIS	none
ADI	none
median effective dose (mg/day)	
oral	9.50
inhalation	2.70
Carcinogenic effects	
Carcinogenic Potency Factor (1.0E-06 cancer risk)	
oral 1/(mg/kg/day)	1.10E-02
inhalation	4.60E-03
10% effective dose(mg/kg/day)	
oral	6.67
inhalation	6.67
Cancer Risk	
Inhalation at 1 ug/m3 (risk)	4.10E-06
water (ug/L,1.0E-06 risk)	2.70E-04
Classification, EPA	B2
Classification, IARC	insufficient evidence

epoxidation oxide and subsequent intramolecular rearrangement. In humans, PCE is metabolized to trichloroethanol, trichloroacetic acid and unidentified chlorinated hydrocarbons. PCE is eliminated primarily through expired air with the metabolites excreted in the urine.

D.5.3 Toxic and Carcinogenic Studies

Tetrachloroethene vapor is a narcotic. Rats did not survive when exposed for longer than 12-18 minutes to 12,000 ppm; when exposed repeatedly to 470 ppm they showed liver and kidney injury. Cardiac arrhythmias attributed to sensitization of the myocardium to epinephrine have been observed with certain other chlorinated hydrocarbons, but exposure of dogs to concentrations of 5000 and 10,000 ppm tetrachloroethene did not produce this phenomenon. Four human subjects were unable to tolerate 5000 ppm in a chamber for 6 minutes. They experienced vertigo, nausea, and mental confusion during the 10 minutes following cessation of exposure. In an industrial exposure to an average concentration of 275 ppm for 3 hours, followed by 1100 ppm for 30 minutes, a worker lost consciousness; there was apparent clinical recovery 1 hour after exposure but the monitored concentration of PCE in the patient's expired air diminished slowly over a 2-week period. Long-term industrial exposures have been reported to cause various neuropathies, such as numbness, trembling, neuritis, and defects of memory. During the second and third post-exposure weeks, the results of liver function tests became abnormal, suggesting that acute exposure had a significant effect upon the liver. Other instances of liver injury following industrial exposure have been reported. Other effects on humans of inhalation of various concentrations are as follows: 2000 ppm, mild narcosis within 5 minutes; 600 ppm, sensation of numbness around the mouth, dizziness, and some incoordination after 10 minutes. In human experiments, 7-hour exposures at 100 ppm resulted in mild irritation of the eyes, nose, and throat; flushing of the face and neck; headache; somnolence; and slurred speech. Exposure of the skin to the liquid for 40 minutes resulted in a progressively severe burning sensation beginning within 5 to 10 minutes; the result was marked erythema, which subsided after 1 to 2 hours. The liquid sprayed into rabbits' eyes produced immediate pain and blepharospasm; patches of epithelium were lost, but the eyes recovered completely within 2 days.

D.5.4 Applicable and Relevant Standards

The recognized applicable and relevant standards for tetrachloroethene are summarized in Table D-5. The ambient water quality criterion for the protection of freshwater life is <5.3 mg/L. A proposed MCLG has been established at 0 mg/L for PCE. Regulations for workplace exposures have been developed by OSHA (100 ppm TWA or 670 mg/m^3) and ACGIH (50 ppm or 335 mg/m^3). EPA has classed tetrachloroethene as a Group B2 compound.

TABLE D-5.

Summary of Toxicological Information for Tetrachloroethene

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA MCL (proposed, mg/L)	none
EPA MCLG (proposed, mg/L)	0
EPA Water Quality Criteria (mg/L)	
fish and drinking water	8.00E-04
fish only	8.85E-03
protection of aquatic life	<5.3
EPA Drinking Water Health Advisories (mg/L)	10kg(70kg)
1 day	none
10 days	34
chronic	1.94(6.8)
OSHA 8 hr TWA ppm(mg/m3)	100 (670)
ACGIH 8 hr TWA ppm(mg/m3)	50 (335)
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	
AIC	0.02
AIS	none
ADI	none
inhalation (mg/kg/day)	
AIC	none
AIS	none
ADI	none
median effective dose (mg/day)	
oral	1.46E+03
inhalation	7.27E+03
Carcinogenic effects	
Carcinogenic Potency Factor (1.0E-06 cancer risk)	
oral 1/(mg/kg/day)	5.10E-02
inhalation	1.70E-03
10% effective dose(mg/kg/day)	
oral	3.23
inhalation	3.23
Cancer Risk	
Inhalation at 1 ug/m3 (risk)	none
water (ug/L,1.0E-06 risk)	8.85E-03
Classification, EPA	B2
Classification, IARC	2B

D.6 Ethylbenzene (EPA 1985f; Mackison et al, 1981)

D.6.1 Summary of Health Effects Data

Ethylbenzene is primarily an irritant of skin and, to some degree, of eyes and upper respiratory tract. Systemic absorption causes depression of the central nervous system with narcosis at very high concentrations. Aspiration of small amounts causes extensive edema and hemorrhage of lung tissue. It is readily metabolized and excreted chiefly as mandelic acid in the urine.

D.6.2 Pharmacokinetics and Metabolism

Ethylbenzene is absorbed through the skin, by inhalation and by ingestion in humans. Absorbed ethylbenzene is distributed throughout the body in rats with the highest levels detected in the kidney, liver, lung, adipose tissue, and digestive tract. In humans, ethylbenzene undergoes rapid metabolism to form primarily mandelic acid and phenylglyoxylic acid. These are not the predominant metabolites formed in animal species. Ethylbenzene is eliminated rapidly primarily through urinary excretion and expired air.

D.6.3 Toxic and Carcinogenic

Wolf et al. exposed guinea pigs, Rhesus monkeys, rabbits, and rats to concentrations of 400 ppm to 2,200 ppm ethylbenzene for 7-8 hrs/day, 5 days/wk, for up to 6 months. No effects were seen in guinea pigs, monkeys or rabbits. In rats, dose levels of 600 ppm and above caused increased liver and kidney weights and histopathological changes in the kidneys and liver, while 400 ppm produced only increased liver and kidney weights.

A critical experiment for calculating a chronic ADI for ethylbenzene is a study of the effects of oral exposure in rats. Rats received ethylbenzene in olive oil by gavage at dose levels of 13.6, 136, 408 or 680 mg/kg/day, 5 days/wk, for 6 months (182 days). Increases in liver and kidney weights as well as slight histopathological changes in these organs were observed at the two highest dose levels. No observable effects were noted in rats exposed to 13.6 or 136 mg/kg/day. Parameters examined included growth, mortality, appearance and behavior, hematology, terminal blood urea nitrogen (BUN) concentration, organ weights, body weight, bone marrow counts, and histopathology.

D.6.4 Applicable and Relevant Standards

The recognized applicable and relevant standards for ethylbenzene are summarized in Table D-6. The ambient water quality criterion for the protection of freshwater life is <32 mg/L. A proposed MCLG has been established at 0.68 mg/L. Regulations for workplace exposures have been developed by OSHA and ACGIH at 100

TABLE D-6.

Summary of Toxicological Information for Ethylbenzene

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA MCL (proposed, mg/L)	none
EPA MCLG (proposed, mg/L)	0.68
EPA Water Quality Criteria (mg/L)	
fish and drinking water	1.40
fish only	3.28
protection of aquatic life	<32
EPA Drinking Water Health Advisories (mg/L)	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA ppm(mg/m3)	100 (435)
ACGIH 8 hr TWA ppm(mg/m3)	100 (435)
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	
AIC	0.1
AIS	0.97
ADI	0.1
inhalation (mg/kg/day)	
AIC	none
AIS	none
ADI	none
median effective dose (mg/day)	
oral	7.24E+02
inhalation	7.24E+02
Carcinogenic effects	
Carcinogenic Potency Factor (1.0E-06 cancer risk)	
oral 1/(mg/kg/day)	none
inhalation	none
10% effective dose(mg/kg/day)	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m3 (risk)	none
water (ug/L,1.0E-06 risk)	1.4
Classification, EPA	noncarcinogen
Classification, IARC	3

ppm or 435 mg/m^3. Ethylbenzene is considered to be a noncarcinogen by EPA.

D.7 Toluene (EPA 1983, 1985f, 1985g)

D.7.1 Summary of Health Effects Data

There is no conclusive evidence that toluene is carcinogenic or mutagenic in animals or humans. Oral administration of toluene at doses as low as 260 mg/kg produced a significant increase in embryotoxic lethality in mice. Decreased fetal weight was observed at doses as low as 434 mg/kg, and increased incidence of cleft palate was seen at doses as low as 867 mg/kg. However, other researchers have reported that toluene is embryotoxic but not teratogenic in laboratory animals. Acute exposure to toluene produces central nervous system depression and narcosis in humans. However, even exposure to quantities sufficient to produce unconsciousness fail to produce residual organ damage. Chronic inhalation exposure to toluene at relatively high concentrations produces cerebral degeneration and an irreversible encephalopathy in mammals. The oral LD_{50} value and inhalation LC_{LO} value for the rat are 5,000 mg/kg and 15,000 mg/m^3, respectively.

D.7.2 Pharmacokinetics and Metabolism

Toluene is rapidly absorbed through the lungs, gastrointestinal tract, and somewhat slower through the skin in both man and rodents. Skin absorption is directly related to the concentration and does appear to be a major route of entry into the body. Little is known about the tissue distribution of toluene in humans, however, based upon its lipophilic nature and low water solubility, toluene would be expected to distribute and accumulate in the lipid tissue (adipose, bone marrow). Toluene distribution in rats is throughout the body, with the greatest accumulation in the lipid tissues. Toluene is metabolized by side-chain hydroxylation to benzyl alcohol, which is conjugated with glycine to form hypuric acid. Minor amounts of toluene undergo ring hydroxylation, probably via arene oxide intermediates, to form o-cresol and p-cresol. In both humans and animals, toluene is excreted rapidly as unchanged compound in expired air. Most of the urinary excretion of toluene occurs within 12 hours of the termination of exposure.

D.7.3 Toxic and Carcinogenic Studies

Toluene does not appear to present carcinogenic risks based upon data available at this time. Exposures to levels of toluene did not produce an increased incidence of neoplastic, proliferative, inflammatory, or degenerative lesions in rats. Toluene does not appear to be carcinogenic when applied to the shaved skin of mice. Toluene has been tested for genotoxicity (mutagenicity) by many investigators using various assay methods and has not been demonstrated to be genotoxic. Investigations into the

teratogenic (reproductive effects) of toluene on mice resulted in statistically significant increases in the incidence of cleft palate. However, no studies have been conducted in humans. The acceptable intakes for chronic and subchronic exposure via the oral route are 3.00×10^{-1} and 4.30×10^{-1} mg/kg/day, respectively. The acceptable intake for both chronic and subchronic exposure via the inhalation route is 1.50×10^{0} mg/kg/day.

D.7.4 Applicable and Relevant Standards

The recognized applicable and relevant standards for toluene are summarized in Table D-7. The ambient water quality criterion for the protection of fresh water life is <17.5 mg/L. An MCLG has been proposed for toluene at 2.0 mg/L. The acceptable intake for chronic exposure via inhalation and oral routes are 1.50×10^{0} and 3.00×10^{-1} mg/kg/day, respectively. The acceptable intakes for subchronic exposure via inhalation and oral routes are 1.50×10^{0} and 4.30×10^{-1} mg/kg/day, respectively. Regulations for workplace exposure developed by OSHA and ACGIH are 100 ppm or 375 mg/m^3. Toluene is considered to be a noncarcinogen by EPA.

D.8 Phenol (Mackison 1981; NIOSH 1976; EPA 1985g)

D.8.1 Summary of Health Effects Data

Phenol appears to have tumor-promoting activity in many strains of mice when repeatedly applied to shaved skin after initiation of known carcinogens. Although there is equivocable evidence that phenol may be weakly carcinogenic when applied to the skin of one sensitive strain of mice, it does not appear to be carcinogenic when applied to the skin of standard strains of mice. NCI reported that phenol was not carcinogenic when administered in drinking water to rats and mice. There is equivocal evidence that phenol may have mutagenic effects although further evaluation is needed. There are no reports of teratogenic effects caused by exposure to phenol.

D.8.2 Pharmacokinetics and Metabolism

The absorption routes for phenol into the human body are inhalation, oral and dermal. Phenol is distributed to tissues (liver, intestines, kidney, spleen, pancreas and extracellular fluid) where metabolism occurs. The primary metabolites of phenol in the body are conjugated phenylglucuronide or phenylsulfuric acid products. These are subsequently oxidized to catechols, quinones, carbon dioxide, and water. Unchanged phenol and its metabolites are excreted in the urine, feces and expired air.

TABLE D-7.

Summary of Toxicological Information for Toluene

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA MCLG (mg/L) (Proposed)	2
EPA Water Quality Criteria (mg/L)	
fish and drinking water	1.43E+01
fish only	4.24E+02
protection of aquatic life	<17.5
EPA Drinking Water Health Advisories (mg/L)	
1 day	18 (10 kg)
10 days	6 (10 kg)
chronic	10.8
OSHA 8 hr TWA (mg/m3)	375(100ppm)
ACGIH 8 hr TWA (mg/m3)	375(100ppm)
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	
AIC	3.00E-01
AIS	4.30E-01
ADI	none
inhalation (mg/kg/day)	
AIC	1.50E+00
AIS	1.50E+00
ADI	none
median effective dose (mg/day)	
oral	2.69E+03
inhalation	2.69E+03
Carcinogenic effects	
Potency Factor (1.0E-06 cancer risk)	none
oral 1/(mg/kg/day)	none
inhalation	none
10% effective dose (mg/kg/day)	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m3 (risk)	none
water (1.0E-06 risk)	none
Classification, EPA	noncarcinogen
Classification, IARC	3

D.8.3 Toxic and Carcinogenic Studies

Subchronic inhalation exposures to phenol is reported to cause liver, kidney, lung and heart damage in guinea pigs. Slight liver and kidney damage was seen in rats exposed by gavage to 100 mg/kg/day for 20 days. The oral and skin LD_{50}s for the rat are 414 and 669 mg/kg, respectively, and the inhalation LC_{50} is 316 mg/m^3. Phenol is an eye, nose and throat irritant and can cause systemic damage to the nervous system in humans following dermal, oral, or inhalation exposure. The acute toxicity of phenol to fresh water species is expressed over a range of 2-3 orders of magnitude. Acute values for fish species range from 5020 ug/L for juvenile rainbow trout to 67,500 ug/L for the fathead minnow.

The compound phenol does not appear to present carcinogenic results based on data available at this time. Some evidence exists that phenol is a weak skin carcinogen in mice. However, there is no evidence that phenols are carcinogenic or mutagenic at low concentrations within physiologic limits. Well-controlled studies on carcinogenic, mutagenic, and tetratogenic studies of phenol are lacking in human and animals. An acceptable daily intake (ADI) of 0.1 mg/kg/day has been established.

D.8.4 Applicable and Relevant Standards

The recognized applicable and relevant standards for phenol are summarized in Table D-8. The ambient water quality criterion for the protection of fresh water life is <10.2 mg/L. The EPA Office of Solid Waste established the Health Based Number for phenol to be 3.5 mg/L. An acceptable daily intake (ADI) for the noncarcinogenic effects for phenol has been established at 0.1 mg/kg/day. Regulations for work place exposure have been developed by OSHA and ACGIH at 5 ppm (20 mg/m^3). Phenol is considered to be a noncarcinogen by EPA.

D.9 Polychlorinated biphenyls - PCB (Safe,S.,1983; D'Itri,F.M., 1983; USEPA 1985f, 1985g Mackison, et al 1981)

D.9.1 Summary of Health Effects Data

Humans exposed to PCBs (in the workplace or via accidental contamination of food) reported adverse effects including chloracne (a long-lasting, disfiguring skin disease), impairment of liver function, a variety of neurobehavioral and affective symptoms, menstrual disorders, minor birth abnormalities, and probably increased incidence of cancer. Animals experimentally exposed to PCBs have shown most of the same symptoms, as well as impaired reproduction; pathological changes in the liver, stomach, skin, and other organs; and suppression of immunological functions. PCBs are carcinogenic in rats and mice and, in appropriate circumstances, enhance the effects of other carcinogens. Reproductive and neurobiological effects of PCBs

TABLE D-8.

Summary of Toxicological Information for Phenol

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA Health Based Number-EPA Office of Solid Waste(mg/L)	3.5
EPA Water Quality Criteria(mg/L)	
fish and drinking water	3.50
fish only	769
protection of aquatic life	10.2
EPA Drinking Water Health Advisories(mg/L)	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA(mg/m3) (skin)	20 (5 ppm)
ACGIH 8 hr TWA(mg/m3)	20 (5 ppm)
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	
AIC	1.00E-01
AIS	1.00E-01
ADI	1.00E-01
inhalation (mg/kg/day)	
AIC	1.90E-01
AIS	2.00E-02
median effective dose(mg/day)	
oral	5.98E+01
inhalation	8.02E+01
Carcinogenic effects	
Potency Factor (1.0E-06 risk)	
oral (1/mg/kg/day)	none
inhalation	none
10% effective dose (mg/kg/day)	
oral	none
inhalation	none
Classification, EPA	noncarcinogen
Classification, IARC	no ranking

have been reported in rhesus monkeys at the lowest dose level tested, (11 ug/kg body weight/day over several months).

D.9.2 Pharmacokinetics and Metabolism

One of the problems associated with understanding the toxicokinetics of PCBs products is that they are mixtures of many different isomers, each with its own characteristic kinetics of behavior in the animal body. PCBs can be absorbed by dermal or oral contact or by inhalation, although quantitative data seem to be lacking with regard to the latter route of exposure. Dermal absorption of PCB contaminated oils and inhalation of PCBs absorbed onto dust particles are minor routes of absorption and ingestion of PCBs represents the principal mode of entry into the organism. Several studies indicate that PCBs are readily absorbed from the gastrointestinal tract. The rate of metabolic conversion of PCBs is mainly a function of the degree of chlorination, and some isomers are relatively readily metabolized to polar compounds which can be excreted. However, PCB sulphones are formed from some PCB which specifically accumulates in certain tissues, e.g. the lung. A main concern is the high persistence of unchanged bioaccumulated PCBs in fatty tissue from which it is only slowly eliminated.

D.9.3 Toxic and Carcinogenic Effects

Whereas the acute toxicity of the PCBs to mammals is relatively low, a diversity of toxic effects is noted upon chronic exposure at low levels involving several target tissues and organs accompanied by generalized effects like anorexia and weight loss. Notable pathological findings involve the liver (hepatomegaly, fatty liver, necrosis), skin (hyperpigmentation, hyperkeratinization, chloracne), immune system (thymus atrophy, immunosuppression), nervous system (hyperactivity and retarded learning ability in monkeys). The PCBs also induce fetotoxicity in several animal species upon low level administration to the mother (monkeys, 1-5 ppm in diet).

PCBs have been demonstrated to induce liver tumors in rats and mice in some studies and EPA has classified these compounds as Group B2 carcinogens with a potency factor of approximately 4 $(mg/kg/day)^{-1}$. This would place the PCBs among the more potent carcinogens evaluated by the Agency. However, the applicability of the linearized multistage model in this case may be questioned, and the potency factor may represent an appreciable over-estimation of risk. The results from short-term tests have been mainly negative. PCBs have been classified by some authorities as epigentic carcinogens of promoter type.

D.9.4 Applicable and Relevant Standards

The recognized applicable and relevant standards for PCBs are summarized in Table D-9. The ambient water quality criterion for the protection of fresh water life is $<2.0 \times 10^{-3}$ mg/L. A proposed maximum concentration level in drinking water (MCL) has been established at 7.9×10^{-6} mg/L for PCBs. A standard proposed by EPA for PCBs in soils and sediments is 10 mg/L. Maximum limits for residues in foods set by FDA are in the range 0.2 (infant food) to 5 mg/kg (fish and shellfish). Occupational exposure limits have been established at 0.5 mg/m^3 by OSHA and ACGIH. As previously noted, PCBs are considered to be probable human carcinogen by EPA, with calculated lifetime risks of 10^{-5}, 10^{-6}, and 10^{-7}, at 0.79, 0.079, 0.0079 ng/L (drinking water), respectively.

D.10 bis(2-Ethylhexyl)phthalate (Perwak et al.1981, Mackison et al 1981, USEPA 1985g)

D.10.1 Summary of Health Effects Data

bis(2-Ethylhexyl)phthalate is reported to be carcinogenic in rats and mice, causing increased incidence of hepatocellular carcinomas and neoplastic nodules after oral administration. The results of dominant lethal experiments in mice suggests that bis(2-ethylhexyl)phthalate is mutagenic when injected intraperitoneally. However, most experiments conducted with microorganisms and mammalian cells have failed to demonstrate genotoxic activity. Teratogenic and fetotoxic effects have been observed in experimental animals after oral and intraperitoneal administration. Other reproductive effect, including testicular changes in rats and mice, have also been reported.
bis(2-Ethylhexyl)phthalate appears to have a relatively low toxicity in experimental animals. The oral, intraperitoneal, and intravenous LD_{50} values reported for bis(2-ethylhexyl)phthalate in rats are 31 g/kg, 30.7 g/kg, and 0.25 g/kg, respectively. bis(2-Ethylhexyl)phthalate is poorly absorbed through the skin and no irritant response or sensitizing potential from dermal application has been noted in experimental animals or humans. Chronic exposure to relatively high concentrations of bis(2-ethylhexyl)phthalate in the diet have caused retardation of growth and increased liver and kidney weights in experimental animals. Acute median effect values range from 1000 to 11100 ug/L of bis(2-ethylhexyl)phthalate for freshwater cladoceran Daphnia magna. LC_{50} values for the midge, scud, and bluegill all exceeded the highest concentrations tested, which were 1800, 3200, and 7700 ug/L, respectively.

TABLE D-9.

Applicable and Relevant Standards for PCBs

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA MCL(mg/L) (proposed)	7.90E-06
EPA Soil Standard(mg/L) (Proposed)	10
EPA Water Quality Criteria(mg/L)	
fish and drinking water	7.9E-08
fish only	7.9E-08
protection of aquatic life	2.00E-03
EPA Drinking Water Health Advisories(mg/L)	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TLV (mg/m3)	0.5
ACGIH 8 hr TLV (mg/m3,54% Chlorine)	0.5
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	
AIS	none
AIC	none
ADI	none
inhalation(mg/kg/day)	
AIS	none
AIC	none
ADI	none
median effective dose(mg/day)	
oral	none
inhalation	none
Carcinogenic effects	
Carcinogenic Potency Factor (10-6 risk)	
oral 1/(mg/kg/day)	4.34
inhalation	none
10% effective dose(mg/kg/day)	
oral	0.05
inhalation	0.05
Cancer Risk	
Inhalation at 1 ug/m3 (risk)	
water (1.0E-06 risk)	8.00E-08
Classification, EPA	B2
Classification, IARC	2B

D.10.2 Pharmacokinetics and Metabolism

The absorption route of bis(2-ethylhexyl)phthalate to the body is through oral exposure. The most common exposure route for this compound is through blood transfusions. Once in the blood, bis(2-ethylhexyl)phthalate can distribute to the various body organs and tissues and may accumulate in the fatty tissues or the body. Mammalian species can metabolize bis(2-ethylhexyl)phthalate to mono-(2-ethylhexyl)phthalate and subsequently, its corresponding alcohol, ketone, and/or acid. Excretion from the body is through the urine and feces, usually within four to seven days in rodents. Of the absorbed bis(2-ethylhexyl)phthalate less than 3% exists as free phthalic acid.

D.10.3 Toxic and Carcinogenic Studies

bis(2-Ethylhexyl)phthalate is not among the more than chemicals evaluated by the CAG for relative carcinogenic potency as potential human carcinogens. However, it has been investigated by the EPA Office of Research and Development, Environmental Criteria and Assessment Office in Cincinnati, Ohio. A level of evidence in animals indicates that sufficient studies have been conducted to determine the carcinogenicity of bis(2-ethylhexyl)-phthalate. However, inadequate studies have been conducted to determine the level of carcinogenic evidence in humans. EPA has ranked bis(2-ethylhexyl)phthalate as a class B2 or suspected human carcinogen. A carcinogenic potency factor of 6.84×10^{-4} $(mg/kg/day)^{-1}$ for bis(2-ethylhexyl)phthalate was calculated by the EPA. This factor places bis(2-ethylhexyl)phthalate among the least potent of the suspected carcinogens.

D.10.4 Applicable and Relative Standards

The recognized applicable and relative standards for bis(2-ethylhexyl)phthalate are summarized in Table D-10. The ambient water quality criterion for the protection of freshwater life is <0.940 mg/L. A Health Based number from the EPA Office of Solid Waste has been established at 2.0 mg/L for bis(2-ethylhexyl)phthalate. Regulations for work place exposure are 5 mg/m^3 for both OSHA and ACGIH. The CAG has evaluated bis(2-ethylhexyl)phthalate as to its human carcinogenicity (Class B2).

D.11 Di-n-butyl phthalate (EPA 1985g, EPA 1978, EPA 1981, ACGIH 1986)

D.11.1 Summary of Health Effects Data

Swallowing di-n-butyl phthalate may cause nausea, dizziness, light sensitivity, and watering and redness of the eyes. Over-exposure to hot vapors or mists of di-n-butyl phthalate may cause

TABLE D-10.

Summary of Toxicological Information for Bis(2-ethylhexyl)phthalate

Relevant Requirements, Criteria, Advisories or Guidance	Value
Health Based Standard-EPA Office of Solid Waste(mg/L)	2.0
EPA Regulatory Standard(mg/L)	0.700
EPA Water Quality Criteria(mg/L)	
Clean Water Act Water Quality Regulation(mg/L)	
fish and drinking water	15.0
fish only	50.0
protection of aquatic life	<0.940
EPA Drinking Water Health Advisories	
1 day	none
10 days	none
chronic	none
Carcinogenic Potency Factor	
unit risk at (1.0E-06),1/(mg/kg/day)	2.00E-04
OSHA 8 hr TWA (mg/m3)	5
ACGIH 8 hr TWA (mg/m3)	5
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	none
AIC	0.6
ADI	
median effective dose (mg/day)	none
oral	none
inhalation	
Carcinogenic effects	
Carcinogenic Potency Factor (1/(mg/kg/day),1.0E-06 risk)	2.00E-04
oral 1/(mg/kg/day)	6.84E-04
inhalation	none
10% effective dose (mg/kg/day)	
oral	5.00E+01
inhalation	5.00E+01
Cancer Risk	
Inhalation at 1 ug/m3 (risk)	none
water (ug/L,1.0E-06 risk)	none
Classification, EPA	B2
Classification, IARC	no ranking

nose and throat irritation. These are no reports that di-n-butyl phthalate is carcinogenic in animals or humans. Di-n-butyl phthalate has not shown significant positive results of mutagenicity in bacterial test systems. It has been observed that di-n-butyl phthalate causes increased embryo mortality, decreased birth weight, and teratogenic effects in rats, mice and chicks.

The acute toxicity for laboratory animals by most routes of administration is very low. The oral and intraperitoneal LD_{50} values for the rat are of 8.0-10.0 g/kg and 3.05 g/kg, respectively.

D.11.2 Pharmacokinetics and Metabolism

Di-n-butylphthalate can affect the body if it is swallowed, comes in contact with the eyes or skin, or is inhaled as a mist or spray. Once in the blood, di-n-butyl phthalate can distribute to the various body organs and tissues and may accumulate in the fatty tissues of the body. In vitro studies (feeding) with pancreatic lipase indicated that di-n-butyl phthalate metabolism in rats followed a pathway similar to unsaturated fats. However, rats given di-n-butyl phthalate orally, excreted the mono-butyl ester as the primary metabolite in the urine with phthalic acid as a minor matabolite. Excretion from the body is through the urine and feces.

D.11.3 Toxic and Carcinogenic Studies

Extensive experience with dibutyl phthalate as an insect repellant has shown that it is relatively non-irritating to the skin, eyes, and mucous membranes. Aerosols from heated dibutyl phthalate may cause irritation of the eyes and upper respiratory tract. In one report of a human case, accidential ingestion of ten grams of this compound by a chemical operator produced nausea and dizziness with lacrimation, photophobia, and conjunctivitis, but recovery was prompt and uneventful. Animal experiments to determine dermal and oral toxicity of dibutyl phthalate showed that extremely high doses were considered necessary to produce toxic effects. Dibutyl phthalate was found to be teratogenic by intraperitonal injection of doses representing 1/10, 1/5, and 1/3 of the LD_{50} value into female rats at the 5th, 10th, and 15th day of gestation. This probably is of no significance in industrial exposures.

D.11.4 Applicable and Relevant Standards

The recognized applicable and relevant standards for di-n-butyl phthalate are summarized in Table D-11. The ambient water quality criterion for the protection of freshwater life is <0.94 mg/L. Regulations for workplace exposure are 5 mg/m^3 for both

TABLE D-11.

Summary of Toxicological Information for Di-n-butyl Phthalate

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA MCLG (mg/L) (Proposed)	none
EPA Water Quality Criteria (mg/L)	
fish and drinking water	34
fish only	154
protection of aquatic life	<0.940
EPA Drinking Water Health Advisories (mg/L)	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA (mg/m3)	5
ACGIH 8 hr TWA (mg/m3)	5
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	
AIC	0.1
AIS	none
ADI	12.6
inhalation (mg/kg/day)	
AIC	none
AIS	none
ADI	none
median effective dose (mg/day)	
oral	420
inhalation	420
Carcinogenic effects	
Potency Factor (1.0E-06 cancer risk)	none
oral 1/(mg/kg/day)	none
inhalation	none
10% effective dose (mg/kg/day)	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m3 (risk)	none
water (1.0E-06 risk)	none
Classification, EPA	noncarcinogen
Classification, IARC	no ranking

OSHA and ACGIH. The EPA has classified di-n-butyl phthalate as noncarcinogen.

D.12 Diethyl Phthalate (EPA 1985g, EPA 1978, EPA 1981, ACGIH 1986)

D.12.1 Summary of Health Effects Data

There are no reports that diethyl phthalate (DEP) is carcinogenic in animals or humans. However, DEP is reported to be mutagenic in bacterial test systems. Reduced fetal weight, resorptions and dose-related musculoskeletal abnormalities were observed among fetuses from rats exposed intraperitoneally to DEP during gestation.

The acute toxicity for laboratory animals by most routes of administration is very low. Oral, inhalation, and intraperitoneal LD_{50} values of 9,000 mg/kg, 7,510 mg/m^3, and 5,058 mg/kg, respectively, are reported for the rat. The no-effect levels determined from chronic feeding studies of six or more weeks duration are 2,500 mg/kg/day for the rat, and 1,250 mg/kg/day for the dog, with no specific lesion attributable to DEP, and no unusual incidence of tumors. In humans, exposure to heated vapor may produce some transient irritation to the nose, throat, and upper respiratory tract.

D.12.2 Pharmacokinetics and Metabolism

Diethyl phthalate can affect the body if it is swallowed, comes in contact with the eyes or skin, or is inhaled. Once in the blood, diethyl phthalate can distribute to the various body organs and tissues and may accumulate in the fatty tissues of the body. Diethyl phthalate would be expected to metabolize similar to other phthalate esters. The primary metabolite would be monoethylester with phathalic acid as a minor metabolite. Excretion form the body is through the urine and feces.

D.12.3 Toxic and Carcinogenic Studies

In preliminary study of exposure of 150 to 250 workers to vapors in an air mixture of phthalate esters 19 personal air samples (collected in breathing zone of employees), four hours duration each, were taken over eight different days at a number of locations in the vicinity of the operations. The results of the air analysis ranged from 8 to 53 mg/m^3 (1-6 ppm). In a diagnostic multiphasic testing procedure, no phthalates in the blood were found before or after the phthalate exposure and no peripheral polyneurities was observed in the population. Exposure to the heated vapor may produce some transient irritation of the nose and throat, but no reports have appeared that cumulative effects occur in its occupations use. Russian investigators studied working the artificial leather industry in which several phthalate plasticizers were used. Ambient air

concentrations for the plasticizers (mixed esters) varied from 1.7 to 66 mg/m^3. The most frequent complaints were pain, numbness and spasms in the upper and lower extremities. These complaints were related to the duration of exposure and usually began after the 6-7th year of work. Pain and numbness were first noted at rest, frequently at night. This was followed by objective evidence of weakness in the upper and lower extremities. Extensive neurological studies revealed polyneurities in 47 persons (32%) while 49.6% of the workers were classified as essentially healthy. Eighty-one persons were evaluated for disturbance of the vestibular function and 78% showed depression of vestibular receptors. This was the first evidence of neurosomatic dysfunction as was a lowering of the level of the excitability threshold for the ole factory receptors.

No long-term feeding studies, carcinogenicity or reproductive studies conducted with diethyl phthalate are available. The acute toxicity for laboratory animals for diethyl phthalate by most routes of administration is very low, bordering the "relatively harmless" group. EPA and IARC consider diethyl phthalate as a noncarcinogen.

D.12.4 Applicable and Relevant Standards

The recognized applicable and relevant standards for diethyl phthalate are summarized in Table D-12. The ambient water quality criterion for the protection of freshwater life is <52.1 mg/L. Regulations for work place exposure are 5 mg/m^3 for both OSHA and ACGIH. The EPA has classed diethyl phthalate as a noncarcinogen.

D.13 Dimethyl phthalate (EPA 1985g, 1978, 1981; ACGH, 1986)

D.13.1 Summary of Health Effects Data

Dimethyl phthalate (DMP)has been used as an insect repellant in World War II. No skin irritation or sensitization; some skin absorption has been reported. Ingestion causes gastrointestinal irritation and coma, and hypertension has been reported. Like dibutylphthalate, exposure to dimethyl phthalate occurs from spray or mist, rather than from the vapor, unless heat is applied.

D.13.2 Pharmacokinetics and Metabolism

The absorption route of dimethyl phthalate to the body is through oral exposure. Once in the blood, dimethyl phthalate can distribute to the various body organs and tissues and may accumulate in the fatty tissues or the phthalate. Similar to other phthalate esters, the Metabolites would include mono-methyl phthalate and subsequently, its corresponding alcohol, ketone, and/or acid. Excretion from the body is through the urine and feces.

TABLE D-12.

Summary of Toxicological Information for Diethyl Phthalate

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA MCL (ug/L)	none
EPA Water Quality Criteria (mg/L)	
fish and drinking water	3.50E+02
fish only	1.80E+03
protection of aquatic life	<52.1
EPA Drinking Water Health Advisories (mg/L)	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA(mg/m3)	5
ACGIH 8 hr TWA(mg/m3)	5
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	
AIC	13
AIS	none
ADI	438
inhalation (mg/kg/day)	
AIC	none
AIS	none
ADI	none
median effective dose (mg/day)	
oral	29900
inhalation	29900
Carcinogenic effects	
Carcinogenic Potency Factor (1.0E-06 cancer risk)	
oral 1/(mg/kg/day)	none
inhalation	none
10% effective dose (mg/kg/day)	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m3 (risk)	none
water (ug/L,1.0E-06 risk)	350
Classification, EPA	noncarcinogen
Classification, IARC	no ranking

D.13.2 Toxic and Carcinogenic Studies

The acute toxicity of DMP by inhalation is extremely low; the least concentration producing toxic signs and symptoms in the cat is reported to be 10,000 ppm. Intraperitoneally in the mouse the LD_{50} was 3.6 g/kg. Orally there was a wide variation in acute lethality, the LD_{50}s for the rabbit, guinea pig, and the rat were respectively, 1.0, 2.4 and 6.9 g/kg. The dermal LD_{50} by repeated skin application in the rabbit was greater than 4 ml/kg. No skin irritation or sensitization resulted. The pathology was limited to slight renal changes of uncertain significance. Applied undiluted to the rabbit eye, only slight irritation was noted.

D.13.3 Toxic and Carcinogenic Studies

Extensive experience with dimethylphthalate as an insect repellent has shown that it is relatively nonirritating to the skin, eyes, and mucous membranes. Aerosols from heated dimethylphthalate may cause irritation of the eyes and upper respiratory tract. In one fatal case of suicidal ingestion of a mixture containing dimethylphthalate and ketone peroxides, the principal toxic symptoms were marked esophagitis and gastritis with hemorrhage. Animal experiments to determine dermal and oral toxicity of dimethylphthalate showed that extremely high doses were considered necessary to produce toxic effects. Dimethylphthalate was found to be teratogenic by intraperitoneal injection of doses representing 1/10, 1/5, and 1/3 of the LD_{50} value into female rats at the 5th, 10th, and 15th day of gestation. This probably is of no significance in industrial exposures.

D.13.4 Applicable and Relevant Standards

The recognized applicable and relevant standards for dimethyl phthalate are sumarized in Table D-13. The ambient water quality criterion for the protection of freshwater life is <33 mg/L. Regulations for work place exposure are 5 mg/m^3 for both OSHA and ACGIH. The EPA has classed dimethyl phthalate as a noncarcinogen.

TABLE D-13.

Summary of Toxicological Information for Dimethyl Phthalate

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA MCL(mg/L)	none
EPA Water Quality Criteria(mg/L)	
fish and drinking water	313
fish only	2900
protection of aquatic life	<33
EPA Drinking Water Health Advisories(mg/L)	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA(mg/m3)	5
ACGIH 8 hr TWA(mg/m3)	5
Noncarcinogenic effects	
risk characterization	
oral (mg/kg/day)	
AIC	
AIS	
ADI	700
inhalation (mg/kg/day)	
AIC	none
AIS	none
ADI	none
median effective dose(mg/day)	
oral	none
inhalation	none
Carcinogenic effects	
Potency Factor (1.0E-06 risk)	
oral (1/mg/kg/day)	none
inhalation	none
10% effective dose (mg/kg/day)	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m3 (risk)	none
Water (1.0E-06 risk)	313
Classification, EPA	noncarcinogen
Classification, IARC	no ranking